BACTERIOLOGICAL PROFILE OF DIABETIC FOOT AMONG THE PATIENT ATTENDING TERTIARY CARE HOSPITAL IN KULASEKHARAM

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CERTIFICATE

This is to certify that the dissertation entitled "**BACTERIOLOGICAL PROFILE OF DIABETIC FOOT AMONG THE PATIENT ATTENDING TERTIARY CARE HOSPITAL IN KULASEKHARAM**" is a bonafide work done by **Dr.K.Greesh**, **Sree Mookambika Institute of Medical Sciences, Kulasekharam** in partial fulfilment of the University rules and regulations for the award of **M.D** in **Microbiology** (Branch IV) of the TamilNadu Dr.M.G.R Medical University.

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I Solemnly declare that the dissertation **'BACTERIOLOGICAL PROFILE OF DIABETIC FOOT AMONG THE PATIENT ATTENDING TERTIARY CARE HOSPITAL IN KULASEKHARAM'** was prepared by me at Sree Mookambika Institute of Medical Sciences, Kulasekharam under the guidance and supervision of **Dr.B.L.Umapathy.BSc.M.D**, Professor, Department of Microbiology, Sree Mookambika Institute of Medical Sciences, Kulasekharam. This dissertation is submitted to **The Tamilnadu Dr. M.G.R Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D (Microbiology).**

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CONTENTS

BACTERIOLOGICAL PROFILE OF DIABETIC FOOT AMONG THE PATIENT ATTENDING TERTIARY CARE HOSPITAL IN KULASEKHARAM.

ABSTRACT

Introduction: Diabetic foot is a major medical problem worldwide leading to disability.

Aims ad objectives: To determine the bacterial profiles of infected diabetic foot ulcers and the antibiotic sensitivity pattern of the isolates.

Materials and Methods: Samples were collected from 75 patients with diabetic foot ulcers by using sterile swabs, aspirated pus and debridement tissue and they were processed.

Results: The age group of these patients ranged from 41 to 90 years and the maximum number of patients was in the age group of 51 to 70 years. Gram negative bacilli were more predominant (73.8%) and the Gram positive cocci (26.2%). Out of 107 isolates, 70 Gram negative bacilli and 20 gram positive cocci were isolated in the age group of 51 to 70. Pseudomonas species was the predominant isolate followed by Klebsiella species, Proteus species, E.coli, Citrobacter species, Acinetobacter species and Enterobacter species. Among the Gram positive cocci isolated in this age group (51 to 70years) Staphylococcus aureus was predominant. In most of the infections in the age group between 51 to 70 years was polymicrobial (31 cases). Gram negative and Gram positive organisms were highly sensitive to Netilmicin (76% and 81%). Sensitivity to Amikacin was (59% and 73%). Extended Spectrum Beta Lactamase (ESBL) producing organisms were mainly seen in E.coli (67%), Klebsiella (47%).

Conclusion: Diabetic foot ulcer infection should be treated according to culture and sensitivity report. Diabetic foot ulcer treatment should be based on Multidisciplinary approach.

Key words: Extended Spectrum Beta Lactamase, Diabetic foot, Methicillin Resistant Staphylococcus aureus, Coagulase negative Staphylococcus aureus.

INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia and about 150- 170 million people are suffering worldwide from this diseases, as per WHO reports the prevalence of diabetes will be double by 2025. Diabetes mellitus is a worldwide phenomenon, type 2 diabetes is the most common form of diabetes in developing countries like India, hence called diabetic capital of the world. In India prevalence of diabetes in rural population is about 2.4 %, and in urban population is about 4-11.6 %. Complications of diabetes mellitus are peripheral vascular disease, cardiovascular disease, nephropathy, retinopathy, neurological and infections. Uncontrolled hyperglycemia, atherosclerotic vascular disease, sensory neuropathy are the most important risk factors developing diabetic foot ulcer .[1]

Twenty five percent diabetic patients have a risk of developing foot ulcer and limp amputation was 15- 45% higher than non diabetic ulcer.[2]

PATHOGENESIS:

For development of diabetic foot ulcer, the most important risk factors are peripheral neuropathy and impaired blood circulation from peripheral vascular disease

Neuropathy:

Development of neuropathy is as a result of hyperglycemia induced metobolic disorder. The most important one is polyol pathway. Hyperglycemia state willfavour aldose reductase and sorbitol dehydrogenase which will convert intracellular glucose to sorbitol and fructose and due to the accumulation of these sugar products leads to decrease in the synthesis of myoinositol, which is needed for normal neuron conduction . The conversion of glucose leads to depletion of nicotinamide adenine dinucleotide phosphate which is required for detoxification of reactive oxygen species and for synthesis of vasodilator nitric oxide. This leads to oxidative stress on nerve cells and increase vasoconstricton leads to is chaemia, which will result in nerve cell injury and death. This also contributes to abnormal glycation of nerve cells and leads to inappropriate action of protein kinase C and leads to further nerve damage.

In diabetic patients neuropathy develops in motor ,sensory ,autonomic components of nervous system. Imbalance between flexion and extension due to damage of innervations of intrinsic foot muscles ,leads to foot deformities that create abnormal bony prominence and pressure points ,which favour for skin break down and ulceration.

Autonomic neuropathy leads to suppression of the function of sweat and oil gland. Foot loses natural function of the moisturising the skin and becomes dry which leads to breakdown and gradually develops infection.

Sensory neuropathy wounds are unnoticed by the patients which worsenes and exacerbates the development of ulcer.

Vascular disease:

The persistent hyperglycemic state leads to endothelial cell dysfunction and smooth cell abnormalities in peripheral arteries which result in the decrease of endothelium derived vasodilator that leads to vasoconstriction . The diabetes hyperglycemic state leads to increase in thromboxane A2, a vasoconstrictor, platelet aggregation which promote the risk for hyper coagulability, and alteration in the vascular extra cellular matrix leads arterial lumen stenosis. The other factors like smoking, hypertension , hyperlipidemia contribute to the development of peripheral arterial disease.[3]

Pathophysiology: In diabetes patients altered protein and lipid metabolism leads to defective wound healing process. Increased glucose level in the body end up in uncontrolled covalent bonding aldose sugars to a lipid or protein without any normal glycosylation enzyme. This product accumulate on surface of cell membranes, Circulating proteins and structural proteins and this product is called as advanced glycation end product (AGE). Formation of

AGE products occurs on extra cellular matrix protein with slow turn over rate. AGE products alter the properties of matrix protein such as laminin, collagen and vitronectin through intermolecular cross linking or covalent bonding of AGE products. AGE products also cross link elastin type on collagen that leads to increased stiffness and increased synthesis of type III collagen that forms the granulation tissue. AGE on laminin result in reduced binding to type IV collagen in the basement membrane, reduced binding of heparin sulphate proteoglyconand polymer elongation.

Nitric oxide is a stimulator of cell maturation, proliferation and differentiation and increases fibroblast proliferation and thereby collagen production in wound healing. Nitric oxide and L-arginine are needed for proper cross linking of collagen fibres via prolene to maximize the tensile strength and minimise the scaring of healed tissues. Pulsatile flow of blood through vessels will activate endothelial cell specific nitric oxide synthase (EcNOS). Nitric oxide is produced by endothelial cell specific nitric oxide synthase, maintains the proper blood flow to tissues and diameter of blood vessels and also regulates angiogenesis, which plays an important role in wound healing. The diabetic patients reduced ability to produce nitric oxide from L-arginine, due to high glucose associated kidney dysfuction ketoacidosis. results in accumulation of nitric oxide synthase inhibitor that leads to reduced production of nitric oxide synthase and pH dependent nature of nitric oxide synthase.[4]

Diabetic ulcer fibroblasts show various morphological differences, that usually large and widely spread in the culture flask, compared to the spindle shaped morphology of the fibroblast in the age matched controls. They often show numerous vesicular bodies, dialated endoplasmic reticulum and lack of micro tubular structure. Interpretations of this observations would be that inspite of high protein turn over and production in diabetic ulcer fibroblasts, vesicles containing secretory proteins could not travel along the micro tubules to release the products outside. Diabetic ulcer fibroblasts exhibit proliferative impairment that contributes to a decreased production of extracellular matrix proteins and delayed wound contraction and impaired wound healing. For wound healing, extra cellular matrix not only needs to be lay down and also able to undergo remodeling and degradation to develop a mature tissue with appropriate tensile strength.[5]

Past twenty years major increase in mortality among the diabetic people is considered to be due to micro and macro vascular complication. Wound healing process is a step wise repair of last extra cellular matrix (ECM) that develop the largest component of the dermal skin layer. To avoid over or under healing, accurate control and rebuilding is essential ortherwise it may lead to various abnormalities. But in some situation the physiological insult and certain metabolic disorders that impedes the normal steps of the wound healing mechanism. One of the examples of metabolic disorder is diabetes mellitus. Many histopathological studies have shown the prolonged

inflammatory phase in diabetic wound ulcer which leads to delay in the formation of mature granulation tissue and reduction in wound tensile strength. Non healing chronic diabetic ulcers are often treated with extracellular matrix replacement therapy, advanced moist wound therapy, negative pressure wound therapy, bio engineered skin or tissue substitute, growth factors. No therapy is completely perfect as each have their own disadvantages. Moist wound therapy stimulates keratinocyte and proliferation and migration, early angiogenesis, collagen synthesis and wound contraction. Various categories of moist dressings are adhesive, backing film, silicon coated foam, hydrocolloids and hydro gels etc. Moist wound dressing is not the best for exudative wounds because it causes fluid retention. In diabetic ulcer treatment various tissue engineering technologies have come up with a cellular or cellular skin replacement products. New therapies such as platelet rich fibrin wound patch which is effective in chronic diabetic ulcer.[6]

Assessment of diabetic foot ulcers

In 2008 American Diabetes Association of the foot care interested group recommended components of foot examinations for patients with diabetes;

1. Visual inspection of bare foot should be performed in a well – lit room .
2. Should include assessment of shoes – inappropriate foot wear leads to ulcer .

3. Should check between the toes for the presence of ulceration or signs of infection .
4. The presence of callus or nail abnormality should be noted .
5. Temperature difference between feet – suggestive of vascular disease.
6. The foot should also be examined for deformities .
7. To palpate the dorsalispedis and posterior tibial pulses and characterized as present or absent to asses vascular disease .

Wagner ulcer classification :

Grade	Ulcer
0	No open lesion ,skin intact
1	Superficial diabetic ulcer
2	Ulcer involves ligament , tendon ,joint capsule or fascia
3	Grade 2 ulcer with abscess or osteomyelitis
4	Partial fore foot gangrene
5	Extensive foot gangrene.[7]

In diabetes mellitus patients, one of the major complications is diabetic ulcer. Fifteen percent diabetes mellitus patients develop diabetic foot ulcer and leads to 84% of foot amputation.[8]

The present study was carried out to determine the aerobic bacterial isolates cultured from diabetic foot infections and their susceptibility to commonly used antibiotics.

AIMS AND OBJECTIVES

1. To determine the bacterial profiles of infected diabetic foot ulcers and
2. To determine the antibiotic sensitivity pattern of the isolates.

REVIEW OF LITERATURE

In 1973, Burkitt.D.P et al said that diabetes mellitus was a global burden. Key factors for development of diabetes are environmental socio economical and metabolic. Complications of diabetes such as neuropathy is to unbalance glycemia level and is associated with other diseases such as depression and artheroscelerosis.[9]

In 1984 **Sapico F.L** et al said that, diabetic foot infections are treated empirically according to causative microorganisms may improve the patient outcome. When optimal sample collection, transport, and culture techniques are used multiple organisms are isolated from diabetic foot infections. Interactions of organisms within these polymicrobial mixture leads to production of virulence factors such as collagenases, hemolysins , proteases and short chain fatty acids ,that cause inflamation, impede wound healing and leads to chronicity of infection .[10]

In 1992 **Brike J.A** et al said that in india type 2 diabetes is the most common and 31.7million people are diabetics in India. Complications of diabetes include peripheral neuropathy, nephropathy, retinopathy, cardiovascular disease, diabetic foot, hypertension, cerebrovascular disease. In diabetic patients many factors like trauma ,smoking ,durations of diabetes, deformity can cause ulcer in foot ,however neuropathy, peripheral vascular

disease are the two major factors for developing the foot ulcer. Due to these risk factors ,untreated minor abrasions can cause foot ulcer, further can get infection with aerobic and anaerobic bacteria. With the help of broad spectrum antibiotics and proper foot care may help in healing the ulcer .If at any point of delay may lead to complications like amputation of foot or limp.[11]

In 1994 **Caputo.GM** *et al*, observed that in India 33 million people are diabetics which was highest in the world, out of which nearly 15% suffer from the dreaded sequelae of diabetic foot. Certain types of infections are common in diabetics and others more severe. It is not only the numbers that is worrisome, situation is different in India due to socio cultural practices as barefoot walking, religious practices like walking on fire, use of improper foot wear and lack of knowledge about foot care attributes towards increase in the prevalence of diabetic foot. Diabetic foot ulcers are not spontaneous ulcers, but results from the interplay of various factors like peripheral vascular disease, neuropathy, autonomic neuropathy, alteration in the plantar pressure, limited joint mobility and defective foot wear. Cell mediated immunity is mostly affected with abnormalities of polymorphonuclear leukocytes (PMN), monocytes and lymphocytes. There are abnormalities of adherence, chemotaxis, phagocytosis, oxidative burst and intracellular killing, also advanced glycation and products leads to the state of low level persistent activation in polymorphonuclear cells, which leads to spontaneous activation of the oxidative burst and the release of myeloperoxidase, elastase and other

neutrophil granular components which may lead to burn out or tolerant polymorphonuclear leucocytes, also may initiate pathologic process leading to vascular injury. Adapting cellular immunity is also affected with decreased lymphocyte proliferative response.[12]

In 1994, **Foster.AVM** et al, said that in diabetic wound case management, selection of dressing is also important component, specific dressing types could prove beneficial depending on the characteristics of the individual wound. For example saline soaked gauze dressings were inexpensive, well tolerated, atraumatic, moist wound environment. Foam and alignate dressings are highly absorbant and decreasing the risk for maceration in wounds with heavy exudates. However, an ideal dressing should contribute to a moist wound environment, absorb excessive exudates which increase the risk for infections.[13]

In 1995 **Steed D**, discussed the adjunctive wound care treatment were under investigation and in practice for diabetic foot ulcer. With the help of human skin equivalents had been shown to promote wound healing in diabetic ulcers, through the action of cytokines and dermal matrix components , which will stimulate tissue growth and wound closure . A recombinant platelet derived growth factor was also currently in use . Other adjunctive therapies were hyperbaric oxygen therapy (HBOT) and granulocyte colony stimulating factors .HBOT is the delivery of oxygen at higher than atmospheric pressure

to the patients, which leads to increase oxygen concentration level in the blood, increase diffusion capacity to the tissues and also increase partial pressure of oxygen in the tissues, which stimulates neovascularization , fibroblast replication, increase phagocytosis and leukocyte mediated killing of bacterial pathogens in the wound.[14]

In 1995, **Reiber.G E** et al, had detected that lower extremity amputation was the most feared complication of diabetes, in many cases amputation should be a treatment option, with good rehabilitation patient may return to normal activities. However, in countries like Poland the supporting mechanism for amputees are not well developed. Amputation should be considered in very limited situations. One of the indications for amputation is serious infection which could be life threatening sepsis. As reconstruction cannot be performed in ischaemic limb, amputation is considered. And also the amputation is also considered in significant rest pain which was not manageable with analgesics.In a major amputation subsequent outcome of the patient was poor. In 5 years, the mortality rate was as high as 40% to 70%. The multidisciplinary team approach to diabetic foot has been shown as a major reduction in amputation incidence.[15]

In 1995, **Gerding.DN**, said that anerobes are often participate in a mixed infections with aerobes, especially in deep tissue infection. But they are rarely as sole pathogen. Less virulent bacteria coagulase negative

staphylococcus species, enterococcus species or corynebacterium species are also represent as true pathogens.[16]

In 1995, **Armstrong.D.G** et al, said that the role of anerobes is particularly unclear because in many studies, samples were not collected appropreiately for anaerobic culture or due to lack of anaerobic setup in many institutions. Among those who did with appropriate methods, some reports that anaerobes play a minimal role, while others have detected 95% prevalence of anaerobes, in a study with Bacteroidesfragilis being the predominant anaerobe isolated.[17]

In 1996, **Baird.D** et al, identified that diabetic foot ulcer infections are mostly polymicrobial infections, proper management of these infections requires an appropriate antibiotic solution based on the culture and antimicrobial sensitivity testing results.[18]

In 1997, **Stone.J.A** et al reported that uncontrolled diabetes is prone to skin infections, increased blood sugar levels thatleads to inhibit bacteria fighting cell. Skin infections may be hazadours, even small injury may progress to ulcer if not properly treated.[19]

In 1997, **Boyko.E.J** et al, said that hyperglycemia in diabetes mellitus further alters cellular function, damage endothelium of vessel valve and further plaque formation and narrowing of vessels. In people with diabetes,

the effect of atherosclerosis is as high as 2 to 3 times and calf vessels were most affected. Peripheral vascular disease may lead to poor healing and increased risk for amputation. Ischaemia presents as bilateral absence of pedal pulses and claudication pain. To assess the ischaemia, posterior tibial and dorsalispedis arteries pulses should be palpated and also to collect the history of claudication pain. Colour also may be assessed which may be difficult in dark skin, pale on elevation or rubor on dependency may indicate ischaemia. Other characteritics were skin temperature (cold on touch), capillary refill more than six seconds, dry, fissured skin, absence of hair growth, dystrophic toe nails, presence of oedema, pain or dry gangrene. [20]

In 1998, **Smith.D** et al evaluated that diabetic foot ulceration become infected approximately 56%. Signs of infections were cellulitis, increase in local temperature, foul smell, oedema, abscess formation. Due to neuropathy the pain is absent. Leucocytosis, fever may not be present in about 50% diabetic patients. Infection may be caused by gram negative, gram positive bacteria and anaerobes. Short time duration of ulcers were usually infected by single gram positive organism, but chronic ulcers may yield mixed flora, both gram positive and gram negative organisms may be with anaerobes.[21]

In 1998, **Armstrong.D.G** et al, said that risk factors for ulcer development were trauma and pressure. It may be necessary to encourage the no weight strategies such as walker, bed rest, wheel chair and

crutches.Encourage the patient to replace or modify their foot wear. Custom made shoe inserts (orthoses) may be necessary for pressure reduction or redistribution. Selection of devices mustbe taken into consideration that the ability of device to remove pressure, ease of application, cost effectiveness and ability to gain patient compliance. [22]

In 1998 **Reiber G.E** et al suggested platelet rich fibrin therapy for chronic or hard to treat diabetic ulcers. Isolation fibrin or plasma from the patients blood ,which have rich platelets and growth factors to promote natural healing process. Application of these product to diabetic foot ulcers have been shown to accelerate healing. Leucopatch is one such product which is a three layered fibrin patch. It is composed of patients own cells and growth factor ,containing high level of platelets and leukocytes. After six weeks of treatment with Lekopatch application showed significant reduction of wound area (65%).[23]

In 1998, **Pathare.N.A** et al, had described that dibetes mellitus is one of the major health problem in world and in India around eighty million people were diabetic. Asia is contributing more than 60% of worlds population with diabetes. India and China contritubes the largest. Incident of multidrug resistant bacteria has been increased in recent years which leads to increased hospital stay, morbidity, mortality and costs. Diabetic foot infections were polymicrobial, usually mixed organism but the organisms depends on other

factors such as microbial flora of the lower limb, foot hygiene, metabolic factors and use of antibiotics.[24]

In 1998, **Krasner.D** et al proved that neuropathic ulcer, primarily seen on the plantar aspect of the foot at the base of the metatarsal heads and first and fifth base of the toe. Ischaemic ulcer mostly occur as distal lesions on the toe or back of the heal.[25]

In 1998, **Lovery.L.A** et al, said that in diabetic patients, once ulcer has developed, risk of wound progression is increased, that may ultimately leads to amputation in up to 85% patients of diabetic ulceration may require amputation. Team approach to wound care, can prevent at least 40% of amputation in diabetic patients.[26]

In 1999, **Reiber** et al, said that factorswhich may affect wound healing are as follows: wound environment, vascular status, ischaemia, pressure area, glycemic control and nutrition status. Comorbidities, retinopathy, end stage renal disease, hypertension, history of amputation and some medications are also involved in blood glucose control and peripheral vascular disease.[27]

In 1999, **Kelwin.W.S** et al, said that in diabetic foot on average of 5 to 6 strains of organisms are often involved with mixture of aerobic and anaerobic organisms.[28]

In 1999, **Zangaro.G.A** et al, discussed about examination of foot wear, because 55% of traumatic events were result of poorly fitting shoes, health care providers and clients must be able to assess the appropriateness of foot wear. The type of shoe, pattrern of wear,fit,linings,seams,insoles or orthoses and presence of foreign bodies must be assessed and appropriate intervention implemented. When choosing shoes, clients are advised to shop late in the day and to be measured both feet. Shoes should be sufficiently ideal with a deep toe box to accommodate foot changes or deformities. Laces were preferred to accommodate swelling, natural fibers such as soft leather more readily conform to the foot and non skid soles and low heals reduce the risk of falls.[29]

In 1999, **Lipsky. BA** identified that Staphylococcus aureus and beta haemolytic streptococci are the first microorganisms to colonise and infect the skin. Patients with previously treated or with chronic infections, gram negative bacilli mainly Enterobacteriaceae was found. Wounds treated with wet dressing the isolates are specifically Pseudomonas aureginosa.[30]

In 1999, **Tentolouris.N** et al, said that diabetic wound infections caused by Methicillin Resistant Staphylococcus aureus was 30%.[31]

In 2000, **Campbell.LV** et al observed that about 60% of diabetic ulcers were due to neuropathy, about 20% were due to is chaemia and 20% were mixed, Which mean that about 40% of diabetic ulcers have an ischaemic

components which will affect the plan of care. On examination neuropathic foot generally appear as dry, painless, warm, insensate. Ischaemic foot usually cold, atrophic skin, dystrophic nails, absent pulses. Sporadic clauducation may be present. The study stressed to monitor ulcer status and identify change over time, several important characteristics including size, location and presence or absence of infections to be assessed and documented.[32]

In 2000, **Fryberg.R.G** et al, published that charcotarthropathy is a structural abnormality in which joint instability due to muscle and ligament atrophy. Walking a weakened, insensate joint causes structural damage and results in sprains and stress, fractures to the foot. The acute stage presents as inflammatory response with bone resorption and then leads to bone destruction with destruction arch of foot presence as rocket bottom sole and the altered pressure distribution may increase the risk of ulceration.[33]

In 2000, **Sinacore** et al observed that many secondary aging factors also implicated in delay wound healing. In addition, patient education, healing potential, physical environment, wound management and quality of life also are the factors implicated in delayed wound healing. [34]

In 2000, **Sibbalal** et al had detected that intervention to promote wound healing includes control of infection, tissue debridement, avoid further trauma, moist wound environment with proper dressing, oxygenation, perfusion, provision of education, foot wear examination. In tissue debridement, the

purpose is to remove the dead or devitalized tissue. In the management of diabetic foot ulcers, common techniques were used such as mechanical debridement which is irrigation with saline solution, wet to dry dressing. Autolytic debridement (hydro colloids, hydrogels) and surgical debridement, which was the method of choice for wounds with large amount of devitalized tissues or infection, however surgical debridement must be performed only on tissue with adequate blood supply or blood circulation.[35]

In 2000, **Lipsky.B.A** et al, said that several studies have confirmed that, receiving prior antibiotic treatment for chronic infections or lesions were usually polymicrobial.[36]

In 2001, **Calhorn** et al, observed that in controlling infection, intervention depends upon the nature of the infection (acute, chronic and systemic). However general interventions for all wounds promote healing. In addition to debridement, it is necessary to control bacterial balance, support host defense and support medical and pharmacological intervention.[37]

In 2002, **Calhoun** et al, obsevered that in diabetic wound dressing, no one dressing was appropriate for all diabetic wounds or the various stages of healing. Selection of dressing was made on the basis of healing potential and clinical assessment of ongoing wound status. Wound dressing categories include film or transparent, hydrating (hydrocolloids and hydrogels), moisture

retentive (adherent or nonadherent), absorbant (hydrofibres, foam, hypertonic saline, algineter) and antimicrobial (cadixomer iodine, silver agents).[38]

In 2002, **Abbott.C.A** et al proved that 55% of foot ulcers are due to pressure from foot wear which can cause trauma to the foot, with reduced circulation and loss of sensation. Assessment should include checking for callus foot wear and structural abnormality.[39]

In 2003, **Merza.Z** et al, had described that in diabetic foot some of the risk factors such as biomechanical factors, smoking, level of glycemia were strongly associated with environmental factors. In certain societies factors such as average monthly house hold income, racial distribution, education level may contribute to diabetic foot prevalence.[40]

In 2003, **Jeffcoate** et al, discussed about the optimation of wound environment which involves a number of component. This include assessing the wound bed for bacterial balance, exudates and need for debridement. Selection of dressing that can control or manage the wound environment, maintaining a moist wound bed as required, while keeping the surrounding wound skin dry, controlling exudates without dessicating the ulcer bed, eliminate dead space by loosely filling the cavity, ensure that there is adequate pressure relief in the affected area.[41]

In 2004**Anadhi C** et al said that in diabetic foot patient , risk of leg amputation was 15-46 times higher than non diabetic patient . Peripheral neuropathy and poor circulation were the major factors for developing foot ulcer . In diabetic foot infections were usually polymicrobial ,where the milder infections are monomicrobial .[42]

In 2004, **Schultz G.S** etal suggested to evaluate patient wound status and compatibility with treatment goals. The wound edge should be examined to determine the presence of cell migration and wound closure. If wound healing was not occuring, status of those factors could be corrected.[43]

In 2004, **Farish.P.L** et al, said that incase of ischaemic diabetic foot, Bypass surgery is a common method of treatment and reported long term result. Ten years limb salvage rate with surgical bypass of lower limb was upto 90%. In case of multiple occlusion, revascularisation at each point to restore arterial blood flow and increase the chance for limbs salvage. Transluminal angioplasty of the iliac artery in conjunction with surgical bypass in the distal extremity may be implemented and efficiency has been demonstrated in diabetic patients.[44]

In 2004, **RNAO** (Registered nurses association Ontario) conveyed that nurses have a major role to identify the emerging problem, to promote maintainance of healthy feet, advice the client of their risk factors. In literature

it have been identified that five primary risk factors that can be quickly assessed and screened by nurses.

These factors are:

- Circulation.
- Sensation.
- Past history of foot ulcer.
- Structural and biochemical abnormalities.
- Knowledge and selfcarebehaviour.

The presence of one or more of this risk factors is favour for developing foot ulcer and amputation of lower limb. Nurses contribute a key role by identifying such risk factors, informing and providing referrals for clients at risk to prevention strategy.

In 2004, **Lipsky.B.A** et al, reported that in India 40 million people are suffering with diabetes mellitus and of equivalent magnitude in other developing countries. In that upto 20% of patients were struggling with diabetic foot complications and hence are the most commonly faced surgical problem. In treatment was not appropriate, it may lead to amputation or disarticulation of varying levels, atleast once in such patients life time. Most of the diabetic foot infections are initially treated empirically based on the

clinical knowledge of the treating doctor and the prevalence of the microbial pattern in the hospital and locality. It would be prudent if the treatment is directed based on the hierarchy of the organisms most commonly isolated and the most common antibiotic sensitivity pattern of these organisms, at the onset and thus help in a better outcome. Several studies have been conducted world wide with respect to the bacteriology and antibiotic sensitivity pattern. A number of studies have found that Staphylococcus aureus and other gram positive aerobes were the most common causative pathogens, usually isolated in more than 60% of cases.[45]

In 2005, **Koneman.W.K** et al have shown the selection of antibiotic agent for treating diabetic foot infection require the knowledge of the potential microbial pathogen and the resistance to the commonly used antibiotics. To assess the right antibiotic to manage the diabetic foot ulcer infections, the result of misuse and abuse of specific antibiotic studies were needed.[46]

In 2005 **Lipsky.BA** et al ,conducted a multicentre study ('SIDESTEP') showed Ertapenem was ineffective against pseudomonas, compared with Piperacillin / Tazobactam. Although in some wound cultures involved by Pseudomonas aeruginosa, revealed similar outcomes. The authors from this study and several other studies from western countries said that Pseudomonas aeruginosa was the commensal organism rather than a pathogen, since could not require any specific antimicrobial coverage to eradicate Pseudomonas

aeruginosa, need wound care measures, such as avoiding moisture in the periwound area, frequent changing of wound dressing and avoiding hydrotherapy based wound care.[47]

In 2005**, Singh.N** et al said that in India diabetes associated problems are the second most common cause of lower extremity amputation.[48]

In 2005, **Bakker.K** et al, said that globally, every 30 seconds one lower limb is lost due to diabetic foot ulcers.[49]

In 2006, **Kulkarni.J** et al reported that 50% of amputed patients will die within five years of amputation, according to USA data mortality in the group of diabetic foot complications was comparable to the mortality in some types of cancer.[50]

In 2006**, Tsae.S.M** et al, said that Staphylococcus aureus infection in the diabetic foot accentruted the inflammatory process, endothelial injury and blood coagulation, ultimately leading to quicker death.[51]

In 2007 **Citron D .M** et al observed that anaerobes was almost always present in mixed culture . F.magna was predominant organism this was in contrast to other study ,which failed to isolate anaerobic gram positive cocci due to not using selective media for anaerobic gram positive cocci ,suboptimal collecton and transport methods .Most frequently used selective anaerobic medium was brucella agar with laked blood ,kanamycin , and

vancomycinagar, growsB. Fragilis group and Prevotella species ,but not gram positive anaerobes .[52]

In 2007,**Stansbury.L.G** et al described that in diabetic patients foot amputation ratio was higher than soldiers taking active part in military, which incorporate 2.3% of all battle injuries and 7.4% of major limb injuries.[53]

In 2008, **Frykberg.R** et al found that in diabetic and neuropathy patient may develop charcotosteoarthropathy which was characterised by progressive destruction of bones and joints of the diabetic foot accompanying with osteopenia. Incidence of this complication, range between 0.1% to 30%. In about 25% of charcotosteoarthropathy may be missed or diagnoses may be delayed due to lack of specific markers of charcotosteoarthropathy, which may lead to significant deformity, ulceration and amputation of foot.[54]

In 2008 **Flynn.N** et al, said that Staphylococcus aureus is most important pathogen among the staphylococci and found in the environment and anterior nares of 20 to 40% of adults. Other sites of colonisation are axilla, vagina, skin fold and the perineum. Staphycoccusaureus has a variety of virulence factors and the ability to develop and expand resistance to broad spectrum antibiotics. Patients with diabetic foot infection is the major cause of morbidity. They occur in 15% of diabetic patients and 20% of all hospitalized diabetic patients.[55]

In 2009 **Paul S** et al said that foot ulceration and infections are the most frequent serious complication of diabetes mellitus . The annual incidence of leg and foot ulcer was 2.6,5.33, times more common than diabetic coronary disease, stroke, renal failure respectively . About 15% of diabetic patient develop foot ulcer in their life time . Studies past 25 years on bacteriology of diabetic foot infections , but the results are varied and contradictory .[56]

In 2009, **Orji.F.A** et al, have shown that incidence rate was high in males. Polymicrobial organisms (56%) were higher than monomicrobials (53%). Prevalence rate of the bacterial isolates were Clostridium species 51%, Staphylococcus aureus were 60%, E.coli 20% and Klebsiella aerogenes 12%. Antibiotic sensitivity pattern of Clotridium species showed sensitive to Fluoroquinolones and high resistance to beta lactams. E.coli and Klebsiellaaerogenes showed resistant to beta lactams and aminoglycosides. All gram negative organisms showed significant sensitivity to fluroquinolones.[57]

In 2010 **Zubair M** observed that prevalence of diabetic foot ulcer in male was to be 56.6% and female was 30% and ratio of 3.5:1 . bacteriological evaluation of diabetic foot ulcer infections showed that prevalence of gram negative organisms were found to be more than gram positive organisms .The prevalence of multi drug resistant organisms was high in diabetic foot ulcer

patients in india due to misuse of antibiotics , this leads to longer duration of hospital stay and their treatment could be more coastly .[58]

In 2010 **Shakil.S** said that diabetes patients have 10 times higher risk of being hospitalised than non diabetes for soft tissue and bone infections . By the year 2025 in India diabetic populations is expected to increase to 57 million.[59]

In 2011 **Chopdekar K.A** et al did a study on bacteriological profile of diabetic foot infection ,out of 113 samples ,a majority of samples that is 96 (85%) showed polymicrobial growth of which, 29 was mixed growth of only aerobes 67 was mixed growth of aerobes and anaerobes. Out of 290 isolates ,223 were aerobes 67 were anaerobes . Among the aerobes gram negative bacilli were 133 , gram positive cocci were 90 ,majority were Staphylococcus aureus (50) , followed by Pseudomonas aeruginosa (47), Acinetobactor species(2)among the anaerobes ,46 were gram positive cocci, 21 were bacilli in that 16 were gram negative and 5 were gram positive. The majority of anaerobes were Peptostreptococcus species 38(57%) , followed by anaerobic streptococci 8(12%).[60]

In 2011 **Pappu A.K** et al did a study on microbiological profile of diabetic foot ulcer out of 104 samples ,average of 1.08 species per diabetic ulcer patient . In that more than one organism was isolated in only 7.7%. Gram negative aerobes was isolated in 76%, gram positive cocci in 24%. Among the

gram negative aerobes, Pseudomonas was 23% isolated,followed by Klebsiella(17%) , Proteus mirabilis(15%),E.coli (12%), Acinetobactor(6%), Proteus vulgaris (2%) and Streptococci (4%) . Second commonest was Staphylococcus aureus(21%). No anaerobes were isolated .22% of amputees each were infected with Psedomonasaeruginosa and Proteus mirabilis ,18% Klebsiella, 14% Staphylococcus aureus in that 6% were methicillin resistant and Acinetobactor 4% infections in amputees .[61]

In 2012 **Banashankari G.S** observed that choice of specimen for culture and sensitivity was tissue and more specific and sensitive than swab because it yielded pathogenic organism by eliminating contaminants but the same time isolation with swab was reliable ,but there is a possibility of isolating only contaminant, it has to be done with at most care. All ulcers were thoroughly washed with sterile saline to avoid the colonizer rather than pathogenic and specimens were collected by scraping from base of ulcer , wound curettage or aspiration rather than wound swab . Majority of isolation was single organisms it was 64% and rest were polymicrobial and about three or more organisms were 5% .Aerobic facultative organisms were isolated in 98% , anaerobic was 2% . In that 66% gram negative isolates were isolated with predominant organisms being Proteus which was 18% , E.coli were 16% and Pseudomonas 13% . Among the gram positive organisms 19% were Staphylococcusaureus followed by Enterococcus were 9% , coagulase negative Staphylococcus were 5% . Gram negative to gram positive ratio was

1.5:1 . Minimal contaminants and minimal isolation of Staphylococcus epidermidis due to tissue technique method (56%), less use of swab . 18.4% were gram positive organisms,34% were gram negative organisms,remaining had grown both (gram positive and gram negative organisms) .[62]

In 2012 **Esmat M.M** et al observed that diabetes patients have risk of developing diabetic foot ulcer in their life time as high as 25% . 15 to 45 times higher risk of limp amputation in diabetic ulcers than ulcers due to other causes .[2]

In 2012 **Manisha J** et al observed that prevalence of diabetes mellitus depends upon many factors, that is age, sex, socio-economic status, diet, heredity, physicalactivity, life style and environmental factors. Incidence ranges in the annual population from 1.0% to 4.1%, prevalence ranges from 4% to 10% and the life time incidence as high as 25%.Males to females ratio was2.1:1, the mean age of patients was 50.25 $\pm$ 12.5.[63]

In 2012, **Tiwar.S** et al, found that diabetic ulcer with polymicribialinfection had comparatively higher total leucocyte count (16,928$\pm$ 9,642 versus 14,593$\pm$6,687) and haemoglobin level significantly lower (7.9$\pm$2.4 versus 9.2$\pm$ 2.2) than monomicrobial infections. HbA_1C level in both groups were similar (9.9% versus 9.5%).Patient infected with gram negative bacteria were also had significantly lower level of Hb (8.5 $\pm$ 1.9 versus 11.1$\pm$ 2.2), total leucocyte count was higher (16,280$\pm$ 6806 versus

9771+ 3243). Neutrophils level was higher (77 versus 67), than infected with gram positive bacteria.While infected with both gram positive and gram negative bacteria had significantly lower level of Hb (7.6 + 3.2 versus 11.1 + 2.2), than infected only with gram positive microorganism. But this was insignificant when patient infected with only gram negative micro organism. In case of culture negative suspicion of infection was high can use molecular techniques for diagnosis of bacterial infection. To differentiate infected from noninfected foot ulcers, inflammatory markers were used. However positive culture and sensitivity result have a priority over the molecular study for the selection of antibiotics.[64]

In 2012, **Shim.V.R** et al, detected that males were more prone for diabetic foot ulcers than females. This may be due to differences in biomechanics between male and female, specifically high foot pressure decrease joint mobility. Male have nearly twice the odd of having insensate neuropathy as women with diabetes.[65]

In 2012 **McInnes.A.D** et al suggested that diabetic foot protection team working across primary and secondary care could reduce length of hospital stay for diabetic foot ulcer, also reducing major amputation ratio. Median length of hospital stay over the period of three years for diabetic foot ulcer , decreased from 47 days to 19 days.66

In 2012 **Blumberg.S.N** et al suggested that an innovate and promising treatment for diabetic foot ulcer is stem cell theraphy.[67]

In 2012, **Forbes.J** et al, said that, in the treatment of wound amniotic membrane has been used which is rich in collagen and various growth factors and it will support the healing process to improve the wound closure and it will reduce the scar formation. In early they have been used natural amniotic membrane which obtained from labour and delivery. Recently techniques have been developed to dehydrate the material while preserving many of these wound healing attributes to produce temperature stable allograft.[68]

In 2013 **Konar.J** et al reviewed that wound healing was a stepwise repair of lost extra cellular matrix that forms largest component of the dermal skin layer. Accurate and controlled rebuilding is necessary to avoid over or under healing that may lead to various abnormality. Sometimes wound healing was disturbed by certain disorder and physiological insult. One such disorder was diabetes mellitus, disturbs the normal steps of wound healing process. Many histopathological studies show in diabetic wound inflammatory phase will be prolonged, this leads to delay formation of mature granulation tissue and parallel reduction in wound tensile strength. 67cases out of 150 identified bacteriology etiology (38%), single organism was isolated in 58 (87%) among which Pseudomonas aeruginosa was the commonest (21 cases), followed by Escherichia coli (16cases), and Staphylococcus aureus (15 cases), Proteus

vulgaris, Enterococcus, Klebsiella pneumoniae were each 2 cases. Polimicrobial was isolated in 9 cases ,in that Staphylococcus aureus along with Klebsiella oxytoca was isolated in 4cases, rest of 5 cases isolated Pseudomonas aeruginosa with Escherichia coli .Among 50 gram negative bacteria, 23(46%) produced ESBL, 17(33.33%), were Amp C beta lactamase producers and carbapenamase producers were4(8%); 33 gram negative isolates were resistant to fluoroquinolones (66%). Extended spectrum beta lactamase producers were Escherichia coli followed by Pseudomonas aeruginosa. Carbapenamase producers was exclusively Pseudomonas aeruginosa .Amp C beta lactamase producers was Pseudomonas aeruginosa followed by Klebsiella pneumonia andKlebsiellaoxytoca. Out of 19 isolates of Staphylococcus aureus, 7 were methicillin resistant (36.84%). All Staphylococcus aureus were sensitive to both linezolid and vancomycin. One Enterococcus faecium was vancomycin resistant and MIC value was > 64mic.gram/ ml and was sensitive to teicoplanin, Dalfopristin/Quinupristin and Linezolid. Isolated Enterococcus faecium were resistant to Penicillin, Gentamycin, Tetracycline, Fluoroquinolone leaving behind very restricted therapeutic options .[69]

In 2013 **Shanmugam.P** said that poor micro vascular circulation in diabetic ulcer patient, limit the phagocytes and favour the development of infection .Improper foot wear and local injuries further compromise blood circulation in the lower extremities .diabetic foot infections are initially treated empirically , which against known causative organisms improve the outcome.

Many studies in the past 25 years reported on bacteriology of diabetic foot but the results were varied and often contradictory. These could be due to differences in the causative organisms occurred over time, geographical variation, type and severity of infections. Most of the time diabetic foot infections were polymicrobial , proper management of the infections requires an appropriate antibiotic selection based on cultures and antimicrobial susceptibility testing results. In recent years increase in the incidence and prevalence of extended spectrum beta lactamase and carbapenemase producers may be due to paucity of data on extended spectrum beta lactamase and carbapenemaseproducers .[70]

In 2013, **Rani.KL** etal observed that out of 150 cases of diabetic foot 107 were males and 43 females, which mean that diabetic foot infections were common in men than women. This could be because diabetes is more common in men, and are prone for trauma because of their outdoor work. In this study they found that diabetic foot infection were common in 40 to 60 year age group. Out of 150 cases, 98.66% was non insulin dependent diabetes mellitus (NIDDM) type. In this study 53.6% of the cases was suffering from diabetes mellitus for more than 6 years. Enterococcus species was isolated only from 4 cases (1.85%) but in most of other studies they isolated in a range of 4 to 30%. Gram negative bacilli was isolated more commonly in this study which was 188 (87.03%). Among gram negative organism Escherichia coli were the most common 25.46% of the total isolates, followed by Proteus mirabilis

(23.14%) Pseudomonas aeruginosa (13.42%) Klebsiellapneumoniae (12.03%) and the other gram negative bacilli were Proteus vulgaris (6%) Citrobacterfreundii (1.85%), Enterobacter species (3.7%), Morganellamorganii (1.38%).

In the management of diabetic foot infections, extended spectrum cephalosporins group of drugs shows good results. The patients with necrotising infections are severely ill, require broader antibiotic coverage. Sensitivity pattern of Staphycoccusaureus were Vancomycin 100%, Cefotaxime (65.2%), Ceftazidime (56%), Clindamycin (34.78%), Gentamicin (34%) and Ampicillin (34%), Ofloxin (34%) and Amikacin (30.4%). This study showed higher rate of resistance. This due to patients received treatment early which could have eliminated sensitive organisms and remain only resistant organism. Sensitivity pattern of Enterococcus species were Vancomycin (100%), Amikacin (50%), Gentamicin (25%), Netilmicin (25%), Ofloxacin (25%), Cefotaxime (25%), Ceftazidime (25%), Ceftriaxone (25%). Gram negative bacilli is more sensitive to Imepenem, Piperacillin/ Tazobactam, Piperacillin, Amikacin, Cefotaxime, Ceftazidime, Ceftriaxone and Gentamycin, Ofloxacin.

In this study Escherichia coli was the most common isolate (25.46%), which was sensitive to Imepenem (96%), Piperacillin/ Tazobactam (90%), Cefotaxime (76.6%), Piperacillin (72%). Amikacin (65.45%), Ceftriaxone

(54.5%), Ceftazidime (49%), Gentamicin (30.9%) and Ofloxacin (30.9%). Proteus mirabilis sensitive to Imepenem 98%, Piperacillin/ Tazobactum 90%, Cefotaxime 60%, Ceftriaxone 58%, 90% sensitivity to Amikacin. Proteus vulgaris was sensitive to Amikacin 79%, Cefotaxime 38.46% and other routinely used antibiotics showing maximum resistant.

Enterobacter species sensitivity pattern was Ofloxacin (87.5%), Cefotaxime (75%), Amikacin (75%), Ceftazidime (62.5%), Gentamicin (50%), Ceftriaxone (50%). Citrobacterfreundii sensitive to Cefotaxime was 50% and Ceftriaxone 50%. Morganellamorganii was sensitive to Cefotaxime 66%, other antibiotics were 33%. Pseudomonas species was sensitive to Imepenem 93%, Piperacillin/ Tazobactam 83%, Piperacillin 83%, Amikacin was 55%, other routinely used antibiotic were less sensitive.

Patient education was the most important aspect, once patient was diagnosed as diabetes, it is doctors responsibility to advice the foot care in diabetic and make them understand the complications of diabetic foot infection.[71]

In 2013, **Turhan.V** et al reported that in diabetes foot infections Staphylococcus aureus was long been recognized as the predominant micro organism but in this study it was second frequently isolated organisms. In 1990 community acquired MRSA emerged as the important isolates in diabetic foot infections, incorporate between 12 to 40% of all Staphylococcus

species. 44.2% methicillin resistance strain isolated in this study. They found that fusidic acid was effective against all Staphylococcus species, including MRSA and in mild to moderate diabetic foot infection, Fusidic acid may be considered as an important therapeutic alternative.[72]

In 2013, **Salihi.S.A** et al, did a study on 25 patients with diabetic ulcer. In that 17 were non insulin dependent diabetes mellitus, 8 were insulin dependent diabetes mellitus and they belong to age group between 40 to 49 years. This may due to repetitive mechanical force of weight during working. All the 25 samples isolated showed pure form of isolates. Among that 14 (56%) were E.coli, 7 (28%) were Proteus mirabilis and 4 (16%) were Staphylococcus aureus. Antibiotiuc pattern of E.coli was more sensitive to Rifampicin, Ciprofloxacin and Nitrofurantoin. From this study they found that Gentamicin had highest antibacterial activity to E.coli 85.7%, Proteus mirabilis 42.8%, Staphylococcus aureus 75%, while antibiotic resistance of Nalidixic acid to E.coli was 100%, Proteus mirabilis 71.4%. Multidurg resistant may be due to chronic course of admission and chronicity of the wound.[73]

In 2013, **Hameedhefni.A.A** et al, identified that the diabetic wound infections begins superficially, but delay in the treatment and impaired body defence mechanism infection can spread through deeper tissues which leads to complications such as gangrene and amputation.[74]

In 2013, **Jaya.M** et al evaluate that maximum number of patients between the age group of 60 to 65 years. Totally 75 organisms isolated from 50 patients. He isolated monomicrobial and polymicrobial 25% equally. Among the isolates gram negative bacilli was isolated more than gram positive cocci. In gram negative bacilli, Pseudomonas species was higher (16%) followed by E.coli (14.6%) and the other organisms were Methicillin sensitive Staphylococcus aureus (13.3%), Streptococcus pyogens (10.6%), Klebseilla species (8%), Acinetobacter species 8%), Methicillin resistant Staphylococcus aureus (8%), Proteus mirabilis (6.6%), Citrobacter species (5.3%), Enterococcus species (5.3%), coagulase negative Staphylococcus (2.6%), Enterobacter species (1.3%).

Among the gram negative bacilli 37.5% of the isolates were extended spectrum beta lactamase (ESBL) producers, 31% of isolates were Carbapenamase producers. Diabetic foot ulcers were ischaemic 74.1% followed by neuropathic type. Most common locations of diabetic ulcer was toes (big toe followed by second toe) plantar region of the foot and dorsum of foot, which was >70%.[75]

In 2013, **Madanchi.N** et al, did a descriptrive study on 873 diabetic ulcer patients. He found that mean duration of diabetic foot ulcer prior to admission was 79.8 days. Mean haemoglobin A_1C (HbA_1C) level was 9.51% and HbA_1C less than 7% was 14.4%. Previous history of lower limb

amputation was 16.4% of patient. In that 4% of patients had major amputation (above ankle), and 12.5% had minor amputation. Majority of patients had ischaemic type of diabetic foot ulcer was 74%, Neuropathic diabetic foot ulcer was 17.4% and neuroischaemic (both type) was 8.5%. Diabetic foot ulcer in the right lower limb is 53.4%, in the left lower limb 38.8% and both lower limbs are 7.8%. Most common location of diabetic foot ulcer was in big toe. Followed by second toe. Diabetic foot ulcer at the site of previous surgeries like debridement, amputation or venous graft removal for coronary artery bypass graft (CABG).

Out of 873 patients 28.2% of patients underwent lower limb amputation, in that 38.3% was major amputation, 61.7% was minor amputation. Mean duration of hospital stay was 16.7+/- 11.3 days, and mortality rate was 5.2%. Most of the patients developed diabetic foot ulcer in the mean age of 59.3 years. Other studies also found that diabetic foot ulcer to be 55- 60 years. In the study population male patients was dominant, which was 58.1%, Other studies also reported the same, that in the average of 50%-63.3% in their study population.

Most of the patients 85.6% had uncontrolled diabetes mellitus, their HbA_1C level was more than 7%. It is suggesting that diabetic foot ulcers are mostly developed in poorly controlled diabetes mellitus. Due to diabetes mellitus comorbidities 69.6% of cases had preceding episodes of

hospitalization. More than 50% of the patients had renal and cardiovascular, more than 40% had ophthalmic comorbidities.

Diabetic foot ulcer was more common in patients with past history of amputation and foot ulceration which was 57 times higher than patients without this history. 22.4% of patients had previous of hospitalization because of diabetic ulcer. Previous hospitalization due to other comorbidity of diabetes mellitus including renal, cardiovascular, cerebrovascular, ophthalmic complication was 47.2%. Relationship between diabetic foot ulcer and comorbidity was controversial but many reports found that comorbidity is an independent risk factor for development of diabetic foot ulcer and amputation. He found that 74.1% of diabetic foot ulcer was ischaemic type followed by neuropathic type. To compare right lower limb to left lower limb, diabetic foot ulcer by ratio, It was 1.37:1, it is because most patients are right dominant, using more on right which may expose to trauma. Most common locations of diabetic foot ulcer was toes (big toe followed by second toe), plantar region and dorsum of foot, which was more than 70% of the patients in the study. Comparing with proximal part, distal part of limb was more affected. This is mostly because of is chaemia, diabetic neuropathy and trauma. Diabetic foot ulcer at the site of previous surgery was identified in 26 patients, which suggesting to avoid unwanted surgical procedures in left of diabetes mellitus patients. Average stay of hospitalization was 16.7 days and 28.2% of patients needed amputation, major to minor amputation ratio was 1.61:1, amputation

rate will vary from different report in different countries. This study suggested that multi disciplinary approach (by a team of Orthopaedic surgeon, Endocrinologist, Vascular surgeon, Infectionist, Internist, Interventional cardiologist, General practioners, Nurse, Physiotherapist) decreases the amputation rate, because multidisciplinary approach is for early detection, prevention, personal education and multiple therapies (dressing, drainage, frequent debridement, washing along with antimicrobial therapy and daily assessment of wound healing).[76]

In 2013, **Zycover.S** et al, did a randomised, double blind, placebo control study and he indicated that in the treatment of diabetic foot ulcers, soluble beta glucans were effective. Soluble beta glucans are available with the brand name woulganbiogel.[77]

In 2014 **Kaur. N** et al did a study on clinical susceptibility profile of diabetic foot and observed that , out of 106 samples were cultured ,in that 98 was culture positive ,136 organisms was isolated averaging of 1.38 organisms per positive culture. No growth was obtained in 8 patients (7.6%) . Growth of one organisms showed in 70 (71.4%) ,growth of 2 organisms was showed in 18 (18.36%) ,and three or four organisms were isolated in 10 patients (10.2%) . Most commonly isolated organisms were gram negative , which was 67.6%, gram positive organisms were 28.6% ,while yeast were 3.67% . Among the gram negative bacteria , most frequent were Proteus species (27) , followed by

E.coli (25) . Among the gram positive bacteria S.aureus (25) . Most effective antimicrobial agent against gram negative were Meropenem, Polymyxin B, Imipenem, and Piperacillin/Tazobactam. While Linezolid ,Vancomycin , Amikacin were most effective against gram positive organisms.[78]

In 2014, **Kavitha.Y** et al, reported that diabetic population in India about 50.8 million, by 2013 which is expected to increase 87 million. Among the diabetes patients, risk of developing a foot ulcer was 15%. Based on recent studies incidence ranges from 1.0% to 4.1% and the prevalence range from 4% to 10%, suggesting that the life time incidence as high as 25%. In diabetic ulcer patient therapy against known causative organisms may improve the outcome. More than half of the patients who underwent lower limb amputation, will have within five years contralateral amputation. Half of those who underwent amputation will die within three years.

Staphyllococcusaureus, E.coli, Klebseilla species, Proteus species, Pseudomonas species and Enterococcus species are the most frequent isolates from diabetic foot ulcers. The diabetic foot infections were usually polymicrobial due to aerobic, anerobic and Candida species. The severe infections were usually polymicrobial isolates, whereas milder infections are usually monomicrobial. The organisms found in diabetic ulcer infections differ not only patient to patient or hospital to hospital but also from one part of the country to another part of the country.

Diabetic foot ulcer was one the most common complications requiring hospitalization. Male patients are predominant in the study population and maximum number of patients belongs to Wagners grade 3. Diabetic foot ulcers were usually polymicrobial infection, but in this study monomicrobial organisms were predominant. Gram negative bacilli were isolated more than gram positive cocci.

Among the organisms Staphylococcus aureus was predominant (21) of about 32.3% followed by Klebseillapneumoniae(10) 15.38%, Pseudomonas aeruginosa (8) 12.31% but in other studies gram negative organisms were predominent. This could be due to over time, geographical variations and types and severity of infections. Most of the gram positive cocci was highly resistant to Erythromycin, Cephalosporins and Gentamicin. But they were sensitive to Amikacin and Clindamycin. All the gram positive cocci were sensitive to Vancomycin. Most of the gram negative bacilli were found to be highly resistant to Cephalosporins, Ciprofloxacin, Cotrimaxazole and Gentamicin. But they show good sensitivity to Amikacin, Piperacillin/ Tazobactam, Imepenem.

Even if the microorganisms were sensitive to some particular antibiotic, drug is unlikely to reach the therapeutic concentration at the site of infection due to virulence factors such as hemolysins, collagenase, proteases and short chain fatty acids that cause impede wound healing process, inflammation and

chronicity of the infection of wound. In diabetic foot microbial infections were not consistent so that repeated evaluation of microorganisms and their antibiotic sensitivity were necessary for the selection of appropriate antibiotics.[79]

In 2014, **Sudaresh.N.J** et al reported that compare to female, males are predominantly prone to post surgical infections. Most of the patients age group were 51 to 60 (28), followed by 61 to 70 (26) and 41 to 50 (23). Age factors was playing an important role in developing infections and contribute with other factors such as economical status and polypharmacy. Average bacteria per lesion was found to be 1.22. Gram negative organisms were dominated than gram positive and polymicrobial nature were observed. The most frequently isolated organisms were Staphylococcus aureus (39.34%) followed by E.coli (23.77%), Enterococcus (4.91%), Pseudomonas aeruginosa (9.83%), Klebseillapneumoniae (7.37%). Proteus species (6.5%), Acinetobacter species (3.27%), Citrobacter species (1.63%).

Sensitivity pattern among gram positive bacteria were Cefoperazone/Sulbactam (100%), Piperacillin/Tazabactam (100%), Clindamycin (100%), Ofloxacin (91.67%), Rifampacin (93.65%). Resistant pattern among gram negative organisams was Penicillin (8.33%), Cotrimaxazole (0%), Kanamycin (0%) and Erythromycin (6.0%).80

In 2014, **Kamtikar.R** et al did a study on microbiological profile of diabetic foot out of 77 patients, they isolated 104 organism, among that mono microorganism is dominative which was isolated from 68 patients and polymicrobial was isolated in rest of the patients. The study compared with other studies in which they reported that polymicrobials was 64.4% to 83.8%. This difference may be due to local pattern of antibiotic usage, absence of severe and deep wounds, demographic characters and low virulence of microorganisms isolated in this study. In this study, out of 104 microorganism 66 are gram negative organism and is dominative. Among this gram negative organism, Pseudomonas aeruginosa was the most common organism isolated which is about 39 (37.5%). Pseudomonas aeruginosashowed highest resistance to commonly used antibiotics and the highest resistance was seen with Ciprofloxacin , Ofloxacin and levofloxacin with Aztreonam and Imipenem. Other studies also have reported similar findings.[81]

In 2014, **Suganthi.P** et al, published a study on bacteriological profile of diabetic foot. Out of 60 patient, 28 (56%) had monomicrobial which is higher than polymicrobial 22 (44%). From 60 samples, 10 samples did not yield any growth. Among the 50 positive culture gram positive organism were isolated more which was about 51 (63%), gram negative were 30 (37%). Among gram positive organisms Staphylococcus aureus was predominant which was 25% followed by Staphylococcus saprophyticus 15%. Among the gram negative predominant isolate was Pseudomonas aeruginosa (25%)

followed by E.coli 4%. In the antibiotic sensitivity pattern Oxacillin, Vancomycin werethe effective drugs against gram positive organism. Meropenem, Piperacillin, Piperacillin/Tazobactam, Ticarcillin/ Clauvulanic acid, Amikacin were the effective drugs against gram negative organisms. Among the combination antibiotic Piperacillin/ Tazobactam found to be an effective drug of choice.[82]

In 2014, **Sridhar.K** et al, did a study on clinic- bacteriological profile of diabetic foot infections, Out of 66 pseudomonas isolates screened for AMP C beta lactamase, in that 18 were positive, which was 27.2%. Extended spectrum Beta lactamase screened in 59 isolates of Klebsiella species and 31 E.coli, in that 44% in Klebsiella were ESBL producers. And 32.2% E.coli were ESBL producers.[83]

In 2015 **Chavan .S.K** et al said that diabetes mellitus is a chronic disorder and major public health problem in india .Foot ulceration was a most common complication in diabetes mellitus patients ,approximately 15% of during their life time .Amputation of limb has a major impact not only changing the body image , increasing the dependency and cost of treatment . Choosing the empirical antibiotic on diabetic foot ,requires the knowledge about etiologic agents ,improper treatment may develop multi drug resistant . Extended spectrum beta lactamase producing multi drug resistant were reported frequently and 15-30 % diabetic wounds were infected with

methicillin resistant Staphylococcus aureus . Multi drug resistant organism causing infections may increase the hospital stay and cost of management.

Maximum diabetic foot patients were above the age 50 years , age increase incidence of diabetic foot also increase .Majority of diabetic foot patients were diabetes mellitus type 2, comparing with female ,male patients were more that is 76.9% . Majority of ulcers were grade 2 and 3 (53.8%) . most common cause of diabetic foot were noted as neuropathy ,which was 56.4%, mean duration of ulcer was 3 to 180 days .

Out of 78 samples ,76 showed growth ,139 organisms were isolated ,an average of 1.8 organisms per lesion. Monomicrobial organisms were isolated 34 samples which was 44.7%,rest were polymicrobial (55.3%). Most frequently isolated organisms were gram negative 93(69.4%) , followed by gram positive 41 (30.6%) . In gram positive organism Staphylococcus aureus were most commonly isolated, which was 28.4%. Only gram negative organisms were found in 14 cases (18.4%) ,had only gram positive in 35(46%) , remaining had growth of both. 46 (49.5%) isolates of gram negative organisms showed ESBL production, out of 19 isolates of Escherichia coli 13 (68.3%) were ESBL producer, AmpC positive were 4(21.5%) , both ESBL and AmpC were 4(21.5%) .

Out of 28 isolates of Klebsiella, 12(42.9%) were ESBL producer, AmpC positive was 3(10.7%), both were ESBL and AmpC 3(10.7%) . Out of

32 isolates of Pseudomonas aeruginosa 13 (40.6%) were ESBL producer, 7 (21.9%) were MBL positive , both were MBL and ESBL 7%(21.9%) positive . Out of 7 isolates of Acinetobacter species 5 (71.4%) were MBL positive, ESBL were not detected. Out of 38 isolates of Staphylococcus aureus MRSA was seen in 22 (57.9%) .

Among the Staphylococcus aureus, high level resistance to Penicillin, Ampicillin, Erythromycin, Cotrimoxazole, Ciprofloxacin. Maximum sensitivity were seen to Netilmicin, Linezolid .Most of the gram negative organisms were resistant to various classes of antibiotics .Most effective antibiotic against gram negative organisms was Imipenem .

Duration of Diabetes mellitus showed borderline significant association with multi drug resistant organisms infection . Duration of diabetes increases , infection with multi drug resistant organism also increase . No significant association of glycemic control with MDRO and non MDRO infections . Duration of hospital stay increase with multi drug resistant organisms (MDRO) than non multi drug resistant organisms (NMDRO) .Infections with MDRO required significantly more surgical treatment than NMDRO. No significant association of glycemic control with MDRO and NMDRO .[84]

MATERIALS AND METHODS

The study was conducted in a tertiary care hospital at Kulasekharam from June 2014 to August 2015. The study was approved by the Institutional Ethical and Research committee. A total of 75 specimens (pus, swabs, aspirated pus, debridement tissue) were collected from diabetic ulcer patients. The samples were collected in dressing room for out patients and in wards for inpatient and then immediately transported to the laboratory and the specimens were processed without any delay.

Inclusion criteria:

Patient admitted with clinically diagnosed diabetes mellitus, supported by laboratory findings and presented with ulcer.

Exclusion criteria:

- Patient with ulcers, who are not diabetic proved by clinical or laboratory investigations.
- Gestational diabetes mellitus with ulcer.

Sample collection:

The samples were collected according to the grade of ulcer. For superficial ulcer the wound was first rinsed with sterile saline and then pus

samples were collected by sterile cotton swab stick. Two swabs were collected, one for Gram stain and the another for culture. In case of grade 2 or 3diabetic ulcer, the pus was aspirated in a 2ml sterile syringe. If the diabetic ulcer is in grade 4 or 5, the pus was also collected in a 2ml sterile syringe. In case of patients undergoing surgery (debridement, amputations) tissues were collected and the specimens were processed without any delay.[70]

Gram staining:

Materials required for the test-

- Gram stain kit (Hi media), which includes crystal violet, Grams iodine, acetone, safranin.
- Glass slide.
- Bacteriological wire loop.
- Cedar wood oil.
- Light Microscope.

Procedure:

Gram stain was done for all the samples and the procedure handled was as follow:

- Heat fixed smear of specimen stained with crystal violet for one minute.

- Grams iodine poured over the smear for one minute.
- Smear was washed in a running tap water.
- Decolourised with acetone for about 10 to 30 seconds.
- Smear was washed with water and then counter stained with safranin for 30 seconds.
- After drying the slides, a drop of cedar wood oil was placed over the smear and focused under light microscope at 100x oil immersion.[85]

Culture:

Materials required:

- Petri dish.
- Bacteriological wire loop.
- Nutrient agar (Hi media).
- Mac conkey agar (Hi media).
- Blood agar (Hi media).
- Incubator.

Media preparation for 100ml:

Media 100ml.

Nutrient agar 2.8gm.

Mac conkey agar 5.1gm.

Blood agar base 4.25gm.

Nutrient agar (2.8 gm), Mac conkey agar (5.1gm), blood agar base (4.25gm) were mixed in a distilled water respectively and autoclaved at 121^0C for 15 minutes and then medium was cooled to 52^0C to reduce condensation of water on petri dish (For blood agar, 10ml of human blood [10% concentration] was added in the blood agar base medium) and then 14ml were poured in to 90mm of diameter petri dishes, in a sterile laminar air flow cabinet. Dishes were left undisturbed until medium had set, labeled and then stored in refrigerator.[86]

Biochemical media preparation for 20ml:

Media	20ml.
• Peptone	0.3 gm.
• Triple Sugar Iron	1.3 gm.
• Mannitol	0.52 gm
• Citrate	0.48 gm.
• Urease	0.5gm.

Peptone (0.3gm), triple sugar iron (1.3gm), mannitol (0.52gm), citrate (0.48gm), urease (0.5gm) media were mixed in 20ml of distilled water and autoclaved at 121^0C for 15 minutes and then cooled to 52^0C and then poured in the sterile test tubes in an volume of 4 to 5ml. Exceptmannitol, all the other media are slantingly placed and undisturbed until media had set, labeled and then stored in refrigerator.[86]

Procedure:

The swabs/ pus/ grounded tissue were inoculated on Nutrient agar, Mac conkeyagar, blood agar and incubated at 37^0C for 18 to 24 hours.

The isolated colonies was identified by the standard biochemical tests such as:

1. Triple sugar iron agar.
2. Mannitol motility medium.
3. Peptone water for indole production.
4. Christensens urea media for urease test.
5. Simmons citrate medium for citrate utilization test.
6. Following carbohydrates were used for the biochemical test: Glucose, Lactose, Sucrose, Mannitol, Mannose (1% each).
7. Following Deaminase test were carried out to identify the aminoacid fermentation.

Using readymade Hi media kit:

a. Lysine decarboxylase.
b. Arginine dihydrolase
c. Ornithine decarboxylase.
d. Control (without aminoacids).

Procedure in detail:

Two to three similar looking colonies from nutrient agar were picked up with loop and inoculated in the peptone water and then incubated for 2 to 4 hours. And then inoculated into various biochemical test tubes under sterile precautions and then incubated at 37^0C for 18 to 24 hours.[87]

1. Oxidative fermentative test:

Used readymade OF medium (Hi Media), Liquid paraffin. Assessed aerobic, anerobic fermentation.

Procedure:

Tubes of medium are inoculated by stabbing, one tube is covered with layer of liquid paraffin to a depth of 5 to 10mm and both are incubated for upto 30 days.[87]

2. Nitrate reduction test:

Used nitrate broth (Hi Media),

Test reagent :

i) Solution A- Sulphanilic acid

ii) Solution B- Alpha naphthylamine.

Procedure:

Inoculate the medium and incubate for 96 hours then add 0.1ml of solution A and B. The test assessed the reduction of nitrate.[87]

3. Phenylalanine deaminase test:

Assessed the deamination of phenylalanine. Used Phenylalanine medium, 10% ferric choloride.

Procedure:

A medium containing phenylalanine is inoculated with growth of bacterial culture and incubated at 37^0C for over night. A few drops of ferric choloride were added and then observed for any green colour change.[87]

4. Catalase test:

Used 30% H_2O_2 for catalase test.

Procedure:

Four ml to 5ml of 30% hydrogen per oxide was taken in a test tube and then the colonies were picked up with sterile glass rod and inoculated in the hydrogen per oxide and observed for effervescence.[87]

5. Oxidase test:

Used ready made 1% oxidase disc (Hi media) (tetramethyl paraphenylene diamine dihydrochloride).

Procedure:

The discswereallowed to reach room temperature and placed over sterile petri dish and then moistened with distilled water and then colony to be tested was picked with glass rod from nutrient agar and smeared over the moist area. A positive reaction indicated by deep purple colour appearing within 5 to 10 seconds, maximum within 60 seconds.[87]

6. Antibiogram:

i) Materials required for antibiogram:

   a. Muller Hinton agar.
   b. Antibiotic disc.

ii) Media preparation (Muller Hinton agar for 100ml):

Muller Hinton agar (3.8gm) (Hi media) was dissolved in 100ml of distilled water, then autoclaved at 121^0C for 15 minutes and was cooled to 52^0C and then about 14ml was poured in the petri dish in a laminar air floor cabinet. Allowed to set, labeled and then stored in the refrigerator.[86]

iii) Antibiotic disc used:

Disc	Symbol	Microgram/ units

• Amoxicillin	AX	10
• Cefalexin	PR	30
• Cefuroxime	CB	30
• Cotrimoxazole	BA	25
• Ciprofloxacin	RC	5
• Gentamicin	GM	10
• Amikacin	AK	30
• Netilmicin	NT	30
• Ceftazidime	FG	30
• Cefoperazone	CP	75
• Cefotoxime	CF	30
• Piperacillin	PC	100
• Piperacillin/Tazobactam	PT	100/10
• Meropenem	MP	10
• Penicillin	PG	10
• Erythromycin	ER	15
• Clindamycin	CD	2
• Tetracyclin	TE	30
• Vancomycin	VA	30

- Cefoxitin CK 30
- Novobiocin NV 30

iv) Preparation of McFarland standard 0.5 :

0.05ml of 1% barium chloride and 9.95ml of 1% sulphuric acid was taken in a test tube, which corresponding to bacterial concentration of 10^8CFU and sealed and kept in a refrigerator.

v) Procedure:

The organism grown aerobically wasinoculated in the peptone water and incubated at 37^0C for 2 to 4 hours and the turbidity was tested against Mcfarland opacity tube 0.5 and from this growth, lawn culture was made on Muller Hinton agar plate and the appropriate antibiotic discs (based on gram positive, gram negative) were placed and incubated at 37^0C for 18 to 24 hours. The zone size around each antimicrobial disc was interpreted as sensitive or resistant according to CLSI guidelines.[88]

- Phenotypic screening of ESBL:

Materials required for test:

- Muller Hinton agar plate.
- Antibiotic disc:
    - Ceftazidime-30microgram

- Ceftazidime/ Clavulanic acid- 30 microgram/ 10microgram.
- Cefotaxime- 30 microgram.
- Cefotaxime / Clavulanic acid- 30microgram/ 10 microgram.

Procedure:

The organism grown aerobically inoculated in the peptone water and incubated at 37^0C for 2 to 4 hours and the turbidity was tested against Mcfarland opacity tube 0.5 and from this growth lawn culture was made on Muller Hinton agar plate and the both ceftazidime and cefotaxime alone and in combination with clavulanic acid was performed for detection of extended spectrum beta lactamase (ESBL) among the family of Enterobacteriaceae. 5mm or more than that increase in zone of inhibition for either ceftazidime/ clavulanic acid or cefotaxime/ clavulanic acid disc compared to ceftazidimeorcefotaximealonerespectively was taken as a confirmatory evidence of ESBL production. Quality control was performed using Escherichia coli ATCC 25922 for ESBL detection.[2]

- MRSA screening:

Materials required:

- Muller Hinton agar plate.
- Cefoxitin disc (30microgram).

Procedure:

Staphylococcus species were tested for Methicillin resistance with 30 microgram cefoxitin disc by Kirby Bauer disc diffusion method. Quality control was performed using ATCC Staphylococcus aureus 43300 was used for MRSA detection, according to Natinal committee for clinical laboratory standard guidelines (CLSI 2011, volume-31, No-1) a zone of inhibition which was equal to or more than 22mm was considered as susceptible to cefoxitin and the organism was reported as Methicillin Sensitive Staphylococcus aureus. Those isolates produced zone on inhibition was equal of 21mm was considered as Methicillin resistant Staphylococcus aureus (MRSA).[84]

Statistics: The data was collected and entered in a master chart. All the statistical calculations were done through SPSS (Statistical Presentation System Software). Analysis was done using Pearson chi square test, Fishers exact test.

p value <0.05 is significant.

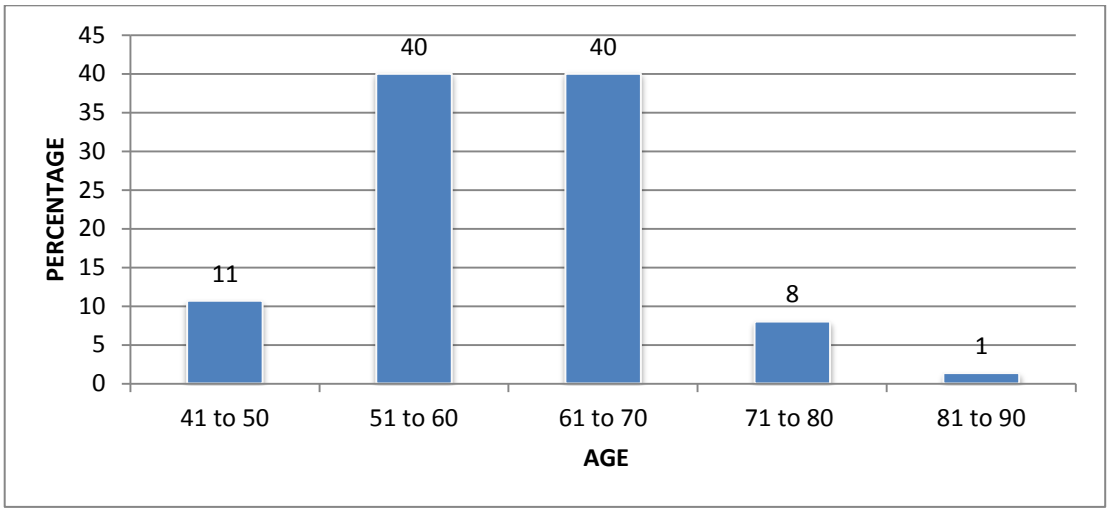
RESULTS

Table: 1 DISTRIBUTION OF AGE

Age	Patients	Percentage (%)
41 to 50	8	11
51 to 60	30	40
61 to 70	30	40
71 to 80	6	8
81 to 90	1	1

The age group varied from 41 to 90 years (Table 1)

Fig .1 Shows Distribution of Age in Percentage



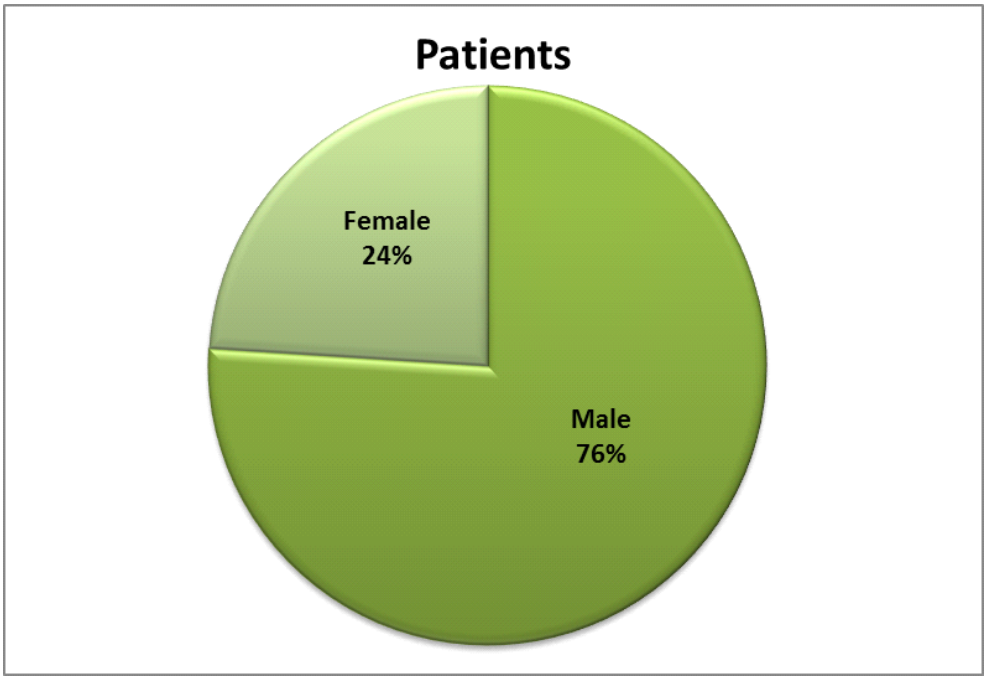
The age group varied from 41 to 90 years (fig:1)

Table: 2 DISTRIBUTION OF SEX

Gender	Patients	Percentage (%)
Male	57	76
Female	18	24

76% were males and 24% were females (Table:2)

Fig .2 Distribution of Sex in Percentage



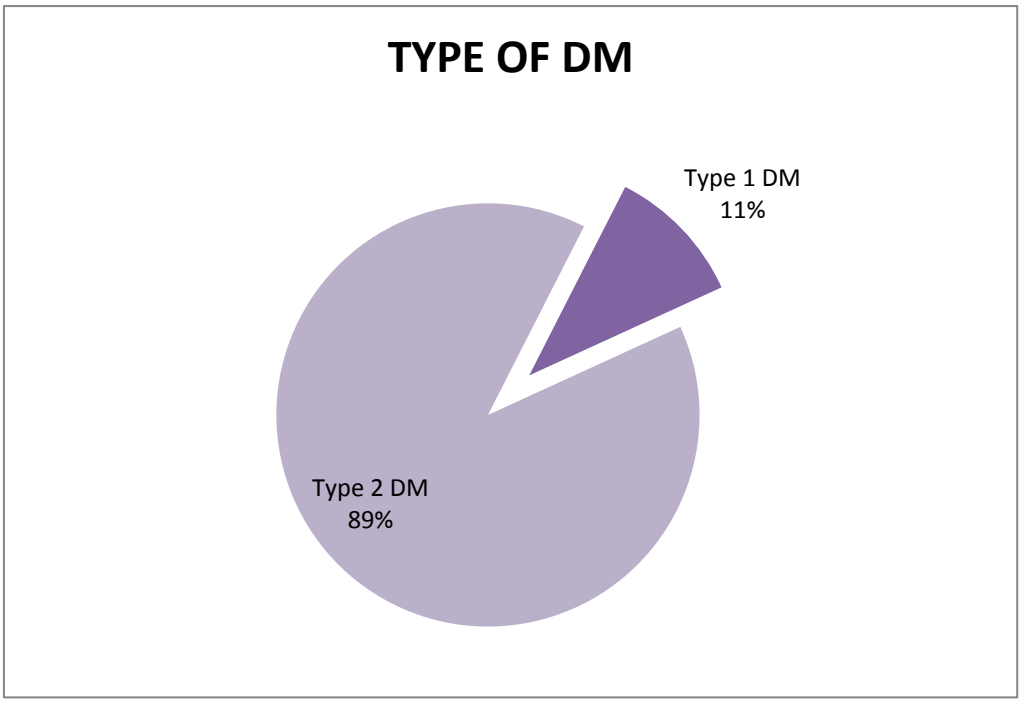
76% were males and 24% were females (fig:2)

Table: 3 TYPES OF DM

DM	Patients
Type 1 DM	8
Type 2 DM	67

Majority of the patients had type II DM (67%) (Table:3)

Fig.3 Type of DM



Majority of the patients had type II DM (67%) (fig:3)

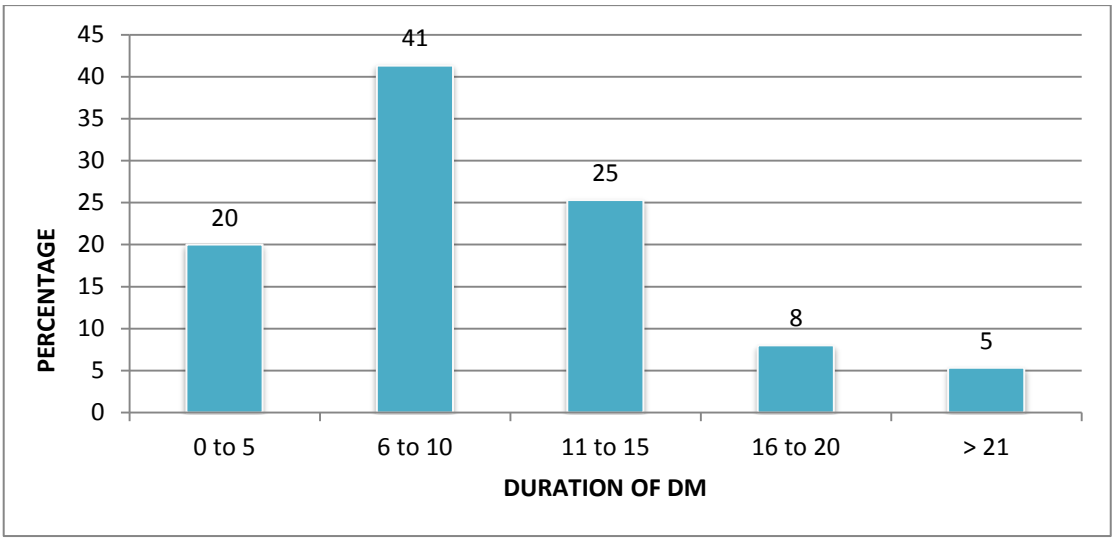
Table:4 DURATION OF DM

DM Duration in years	Patients	Percentage (%)
0 to 5	15	20
6 to 10	31	41
11 to 15	19	25
16 to 20	6	8
> 21	4	5

Duration of DM varied from 6-15 years in a majority of patients studied (66%)

(Table:4)

Fig. 4 Duration of DM



Duration of DM varied from 6-15 years in a majority of patients studied (66%) (fig:4)

Table: 5 HbA_1C LEVEL

DM	HbA1c level	Patients	Percentage (%)
Good control	6.3 to 7.3	10	13
Fair control	7.4 to 7.9	2	3
Poor control	8.0 to 9.3	46	61
Uncontrol	> 9.3	17	23

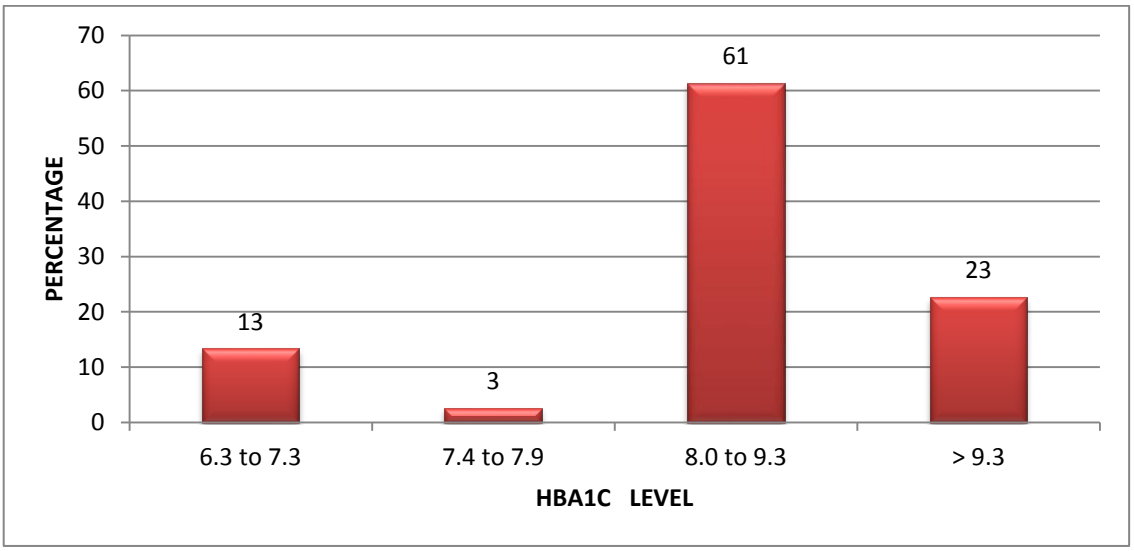
The HbA1C level carried out on the patients showed a very poor control in the majority of patients (61%). Screened which varied from 8 to 9.3%. However the HbA1C level was uncontrollable (more than 9.3) in a large number of patients studied (23%) (Table:5)

Table:5b. Distribution of HbA1C

		Neuropathy	
		Yes	**No**
HbA1C	Fair Control	4	7
	Poor Control	45	19

P value of HbA1C is <0.05. Poor control of HbA1C level was associated with neuropathy, which is statistically significant (Table:5b).

Fig. 5 HbA1C Level



The HbA1C level carried out on the patients showed a very poor control in the majority of patients (61%). Screened which varied from 8 to 9.3%. However the HbA1C level was uncontrollable (more than 9.3) in a large number of patients studied (23%) (fig:5)

Table:6 DISTRIBUTION OF SMOKERS AND NON SMOKERS

Smoking	Patients	Percentage (%)
Yes	53	71
No	22	29

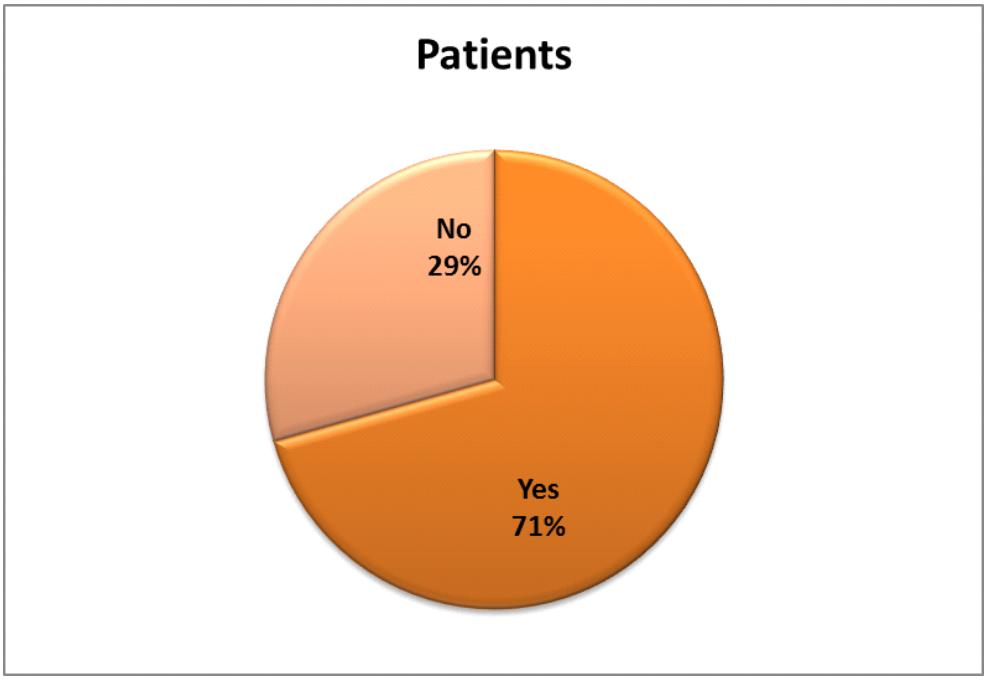
The contributing factor like smoking was seen in 71% of the patients as shown in Table:6.

Table:6(b) DISTRIBUTION OF SMOKING

		Neuropathy	
		Yes	No
Smoking	Yes	39	14
	No	10	12
		Vasculopathy	
		Yes	No
Smoking	Yes	4	49
	No	3	19

p value of smoking is less than 0.05%. Smoking is associated with neuropathy, which is statistically significant.

Fig .6 Smokers & Non - Smokers



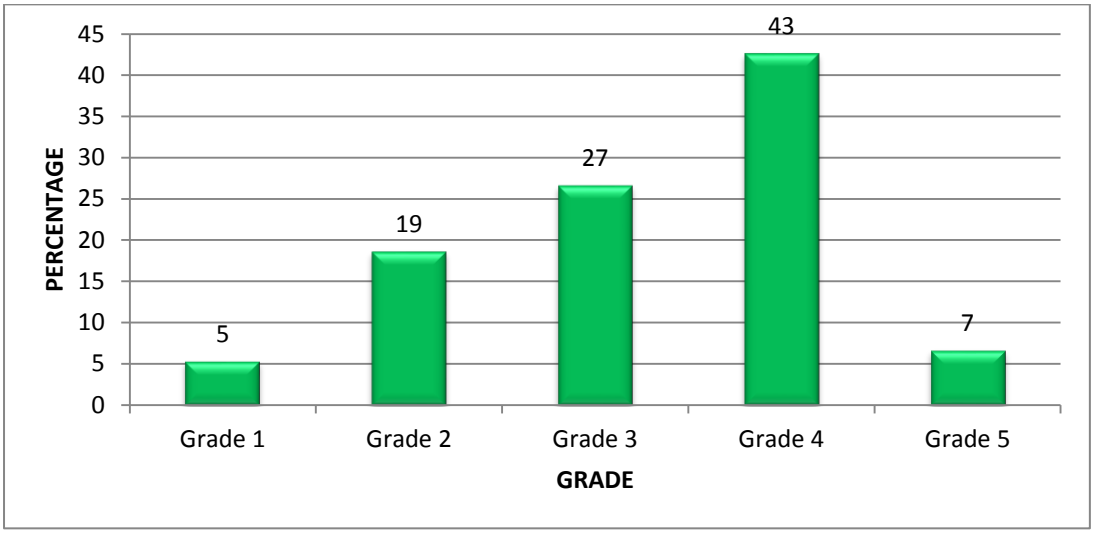
The contributing factor like smoking was seen in 71% of the patients as shown in fig: 6

Table: 7 GRADE OF ULCER

Grade of Ulcer	patients	Percentage
Grade 1	4	5
Grade 2	14	19
Grade 3	20	27
Grade 4	32	43
Grade 5	5	7

Wagners grade 3,4 ulcers was seen in 70% of the studied group as showed in Table:7

Fig.7 Grade of Ulcer.



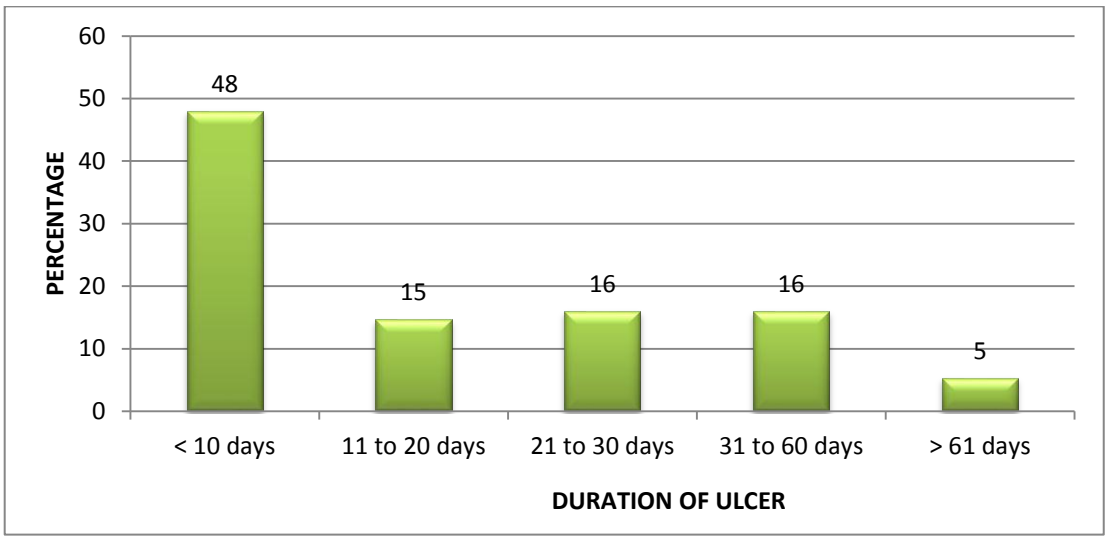
Wagners grade 3,4 ulcers was seen in 70% of the studied group as showed in fig:7

Table: 8 DURATION OF ULCER.

Duration of Ulcer	Patients	Percentage
< 10 days	36	48
11 to 20 days	11	15
21 to 30 days	12	16
31 to 60 days	12	16
> 61 days	4	5

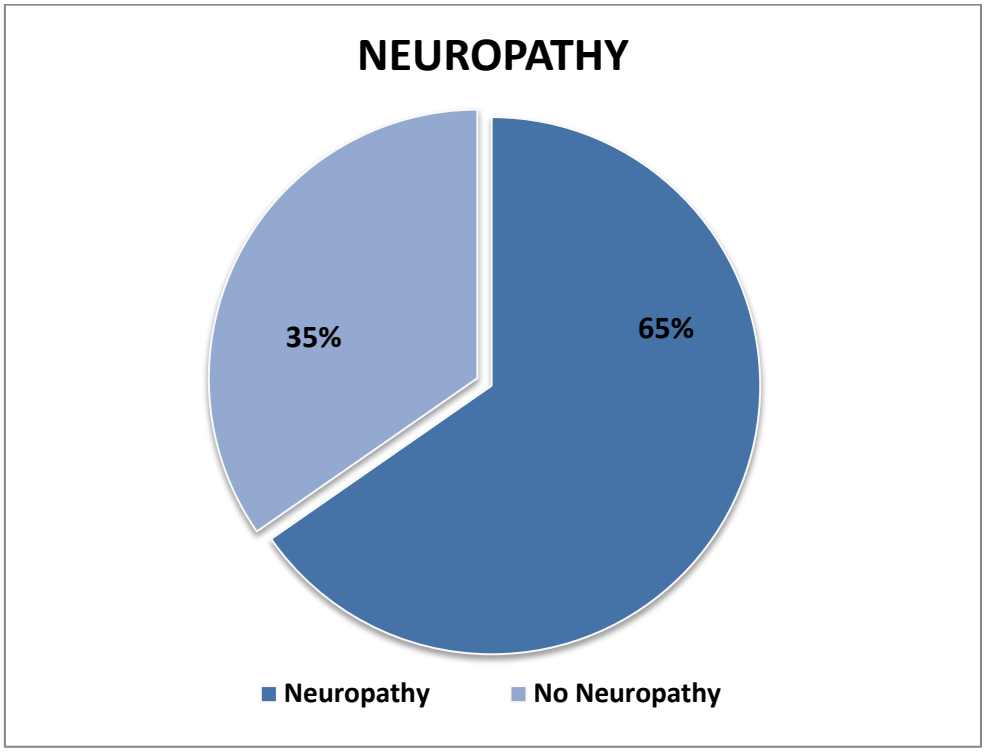
Duration of ulcer was less than 10 days in a majority of patients (48%) as showed in Table:8

Fig. 8 Duration of Ulcer



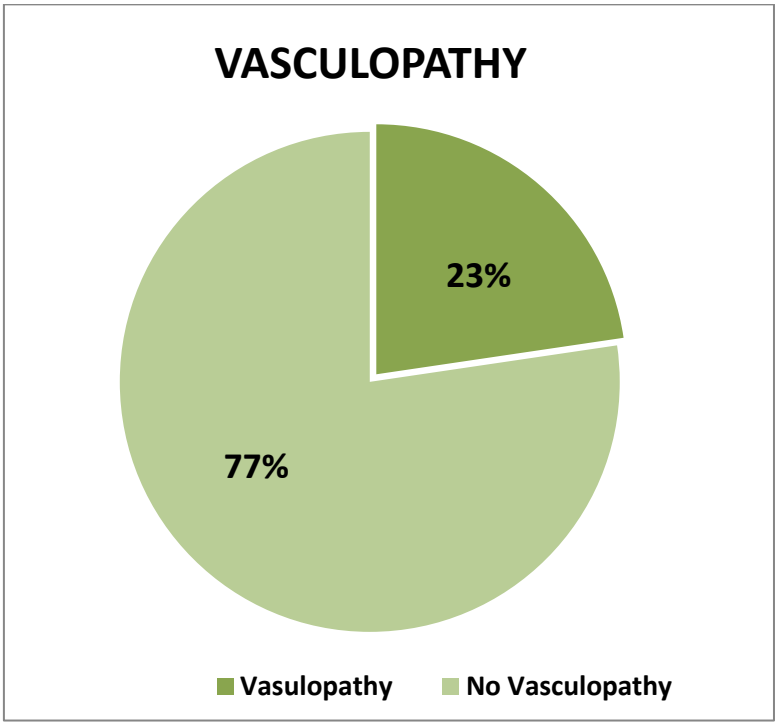
Duration of ulcer was less than 10 days in a majority of patients (48%) as showed in fig:8

Fig.9 Distribution of Neuropathy



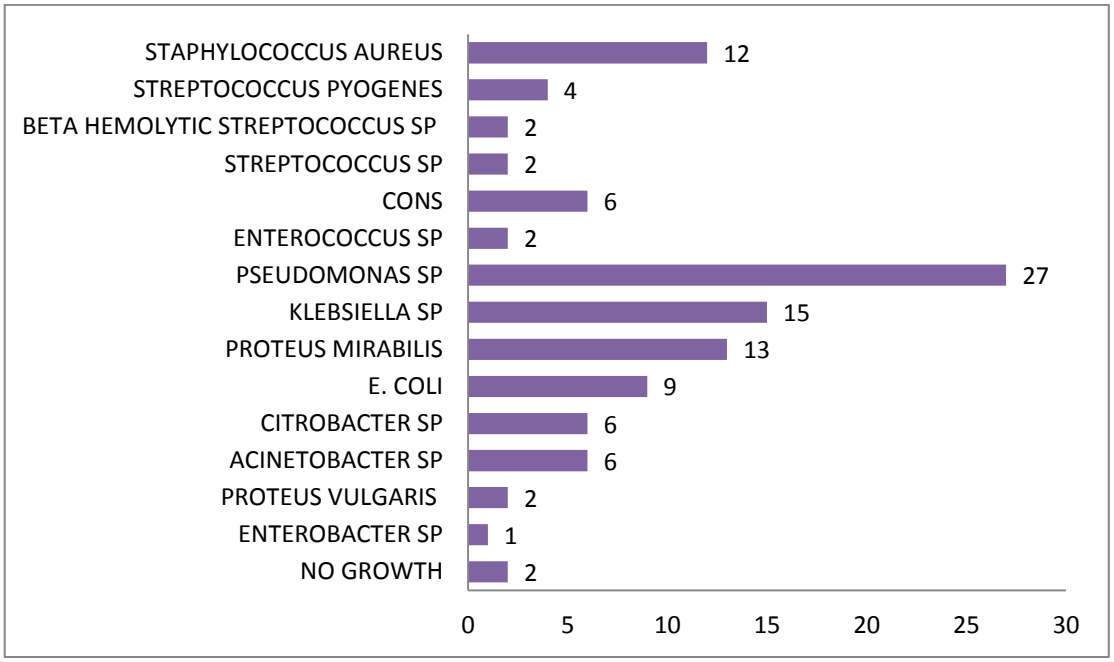
Neuropathy was seen in 65% in the study population (fig:9)

Fig .10 Distribution of Vasculopathy



Vasculopathy was seen in 23% in the study population (fig:10)

Fig. 11 List of Organisms



The predominant pathogens isolated were gram negative bacilli constituting 73.8% and gram positive cocci constituting 26.2%. Among the gram negative bacilli Pseudomonas species was predominant having 36.2% incidence and among the gram positive cocci Staphylococcus aureus were the predominant pathogens accounting 42.8%. (fig:11)

Table:9 ANTIBIOGRAM

	PSEUDOMONAS SP			KLEBSIELLA SP		
	Sensitive	**Intermediate**	**Resistant**	**Sensitive**	**Intermediate**	**Resistant**
PENICILLIN	-	-	-	-	-	-
AMOXYCILLIN	-	-	-	0.00%	0.00%	100%
TETRACYCLINE						
AMIKACIN	52.17%	8.69%	39.13%	69.23%	7.14%	30.76%
PIPERACILLIM/TAZOBACTUM	68.42%	0.00%	31.58%	42.85%	21.42%	35.71%
NETILMICIN	65.83%	15.38%	19.23%	78.57%	0.00%	21.43%
PIPERACILLIN	28.57%	0.00%	71.43%	6.66%	22.66%	60%
CIPROFLOXACIN	23.07%	0.00%	76.92%	25.25%	8.33%	66.66%
COTRIMOXAZOLE				33.33%	0.00%	66.67%
MEROPENEM	53.85%	0.00%	46.15%	90.91%	0.00%	9.09%
GENTAMYCIN	24%	4%	72%	50%	0.00%	50%
VANCOMYCIN	-	-	-			
CEFOLEXIN				20.00%	0.00%	80.00%
CEFUROXIME				23.08%	7.69%	69.23%
CEFOTOXIME	0.00%	0.00%	100.00%	21.43%	0.00%	78.57%
CEFOPERAZONE	30.7%	7.69%	61.5%	20%	13.33%	66.66%
CEFTAZIDIME	33.33%	5.56%	61.11%	20%	0.00%	80%

Seventy four percent of Pseudomonas species was resistant to 3rd generation Cephalosporins. (Table:9)

Table:10 ANTIBIOGRAM

	PROTEUS SPECIES			E.COLI		
	Sensitive	Inter mediate	Resistant	Sensitive	Inter mediate	Resistant
PENICILLIN	-	-	-			
AMOXYCILLIN	21.42%	0.00%	78.57%	11%	0.00%	89.00%
TEICOPLANIN	-	-	-	-	-	-
CLINDAMYCIN	-	-	-	-	-	-
ERYTHROMYCIN	-	-	-	-	-	-
TETRACYCLINE	-	-	-			
AMIKACIN	64.28%	0.00%	35.71%	78%	0.00%	22%
PIPERACILLIM/ TAZOBACTAM	86.66%	13.33%	0.00%	37.50%	0.00%	62.50%
NETILMICIN	63.63%	0.00%	36.36%	86%	0.00%	14%
PIPERACILLIN	63.64%	0.00%	36.36%	11%	0.00%	89%
CIPROFLOXACIN	40%	13%	47.00%	33%	0.00%	67%
COTRIMOXAZOLE	33%	0.00%	67%	50%	-	50%
MEROPENEM	84.62%	7.69%	7.69%	67%	22%	22%
GENTAMICIN	53%	0.00%	47%	67%	0.00%	33%
VANCOMYCIN	-	-	-	-	-	-
CEFOLEXIN	20%	0.00%	80%	22%	0.00%	78%
CEFUROXIME	27%	0.00%	73%	33%	0.00%	67%
CEFOTOXIME	30.77%	7.69%	61.54%	11%	0.00%	89%
CEFOPERAZONE	35.71%	7.14%	57.14%	29%	0.00%	71%
CEFTAZIDIME	53%	13.33%	33%	33%	0.00%	67%

Table 10 shows Proteus species 87% sensitivity to Piperacillin/ Tazobactam, 85% to Meropenem and E.coli shows 89% resistant to Amoxicillin, Piperacillin, Cefotoxime.

Table: 11 ANTIBIOGRAM

	CITROBACTER SPECIES			ENTEROBACTER SPECIES		
	Sensitive	**Intermediate**	**Resistant**	**Sensitive**	**Intermediate**	**Resistant**
AMOXYCILLIN	0.00%	0.00%	100.00%	100.00%		
TETRACYCLINE						
AMIKACIN	100%	0.00%	80.00%			
PIPERACILLIM/ TAZOBACTUM	17%	17%	67%	100.00%		
NETILMICIN	100%	0.00%	0.00%	100.00%		
PIPERACILLIN	20.00%	33%	67%	0.00%	100.00%	
CIPROFLOXACIN	50%	0.00%	50.00%	100.00%		
COTRIMOXAZOLE	83%	0.00%	73%	100.00%		
MEROPENEM	67%	0.00%	33%	100.00%		
GENTAMYCIN	25.00%	25.00%	50.00%	100.00%		
CEFOLEXIN	0.00%	0.00%	100.00%	100.00%		
CEFUROXIME	0.00%	0.00%	100.00%	100.00%		
CEFOTOXIME	0.00%	17%	83.00%	100.00%		
CEFOPERAZONE	17%	17%	67%	100.00%		
CEFTAZIDIME	17%	0.00%	83%	100.00%		

Table 11 shows Citrobacter and Enterobacter species shows 100% sensitivity to Netilmycin

Table:12 ANTIBIOGRAM

	ACINETOBACTER SP		
	Sensitive	**Intermediate**	**Resistant**
AMOXYCILLIN	0.00%	0.00%	100.00%
AMIKACIN	40.00%	0.00%	60.00%
PIPERACILLIM/TAZOBACTAM	0.00%	0.00%	100.00%
NETILMICIN	40.00%	0.00%	60.00%
PIPERACILLIN	0.00%	0.00%	100.00%
CIPROFLOXACIN	0.00%	0.00%	100.00%
COTRIMOXAZOLE	20.00%	40.00%	40.00%
MEROPENEM	0.00%	0.00%	100.00%
GENTAMICIN	0.00%	0.00%	100.00%
CEFOLEXIN	0.00%	0.00%	100.00%
CEFUROXIME	0.00%	0.00%	100.00%
CEFOTOXIME	0.00%	0.00%	100.00%
CEFOPERAZONE	0.00%	0.00%	100.00%
CEFTAZIDIME	0.00%	0.00%	100.00%

Table:12 shows Acinetobacter species 100% resistant to Amoxicillin, Piperacillin/Tazobactam, Piperacillin, Ciprofloxacin, Meropenem, Cefotoxime, Cefuroxime, Cefolexin, Gentamicin.

Table:13 ANTIBIOGRAM

	STAPHYLOCOCCUS AUREUS			CONS		
	Sensitive	**Inter mediate**	**Resistant**	**Sensitive**	**Inter mediate**	**Resistant**
PENICILLIN	9.00%	0.00%	91.00%	0.00%	17.00%	83.00%
TEICOPLANIN	90.00%	10.00%	0.00%	80.00%	0.00%	20.00%
CLINDAMYCIN	91.67%	0.00%	8.33%	60.00%	0.00%	40.00%
ERYTHROMYCIN	15.38%	15.38%	69.23%	0.00%	0.00%	100.00%
TETRACYCLINE	58.00%	17.00%	25.00%	50.00%	0.00%	50.00%
AMIKACIN	63%	25%	12.5%	50.00%	16.67%	33.33%
NETILMICIN	78%	0.00%	22%	60.00%	0.00%	40.00%
CIPROFLOXACIN	33%	0.00%	67%	40.00%	0.00%	60.00%
COTRIMOXAZOLE	18.00%	0.00%	81.00%	33.33%	0.00%	66.67%
GENTAMICIN	40.00%	20.00%	40.00%	75.00%	0.00%	25.00%
VANCOMYCIN	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%
NOVOBIOCIN	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%
CHOLORAM PHENICOL	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%
CEFOXITIN	83.33%	0.00%	16.67%	50.00%	0.00%	50.00%

Eight one percentage of Staphylococcus aureus was resistant to cotrimaxozole in our studyfollowed by Erythromycin (69%), Ciprofloxacin (67%), Gentamicin (40%). (Table:13).

Table:14 ANTIBIOGRAM

	STREPTOCOCCUS PYOGENES			BETA - HEMOLYTIC STREPTOCOCCUS		
	Sensitive	**Inter mediate**	**Resistant**	**Sensitive**	**Inter mediate**	**Resistant**
PENICILLIN	100.00%	0.00%	0.00%	100.00	0.00%	0.00%
TETRACYCLINE	66.67%	0.00%	33.33%	50.00%	0.00%	50.00%
AMIKACIN	75.00%	0.00%	25.00%	100.00%	0.00%	0.00%
NETILMICIN	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%
CIPROFLOXACIN	75.00%	0.00%	25.00%	50.00%	0.00%	50.00%
CEFUROXIME	100%	0.00%	0.00%			
CEFOTOXIME	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%

Streptococcus pygenes and other beta hemolytic streptococci were 100% sensitive to penicillin (100%). However Streptococcus pygenes was also sensitive to Netilmicin, Cefotoxime and Cefuroxime(100%) as showed in Table:14.

Table:15 ANTIBIOGRAM

	ENTEROCOCCUS SP			STREPTOCOCCUS SP		
	Sensitive	Inter mediate	Resistant	Sensitive	Inter mediate	Resistant
PENICILLIN				100.00%		
TETRACYCLINE	50.00%	0.00%	50.00%	50.00%		50.00%
AMIKACIN	50.00%	0.00%	50.00%	100.00%		0.00%
PIPERACILLIM/ TAZOBACTUM	-	-	-			
NETILMICIN	50.00%	0.00%	50.00%	100.00%	0.00%	0.00%
PIPERACILLIN	-	-	-			
CIPROFLOXACIN	100.00%	0.00%	0.00%	50.00%	0.00%	50.00%
COTRIMOXAZOLE	50.00%	0.00%	50.00%	50.00%	0.00%	50.00%
MEROPENEM	-	-	-			
GENTAMYCIN	-	-	-			
VANCOMYCIN	100.00%	0.00%	0.00%	100.00%	0.00%	0.00%
CEFUROXIME	100.00%	0.00%	0.00%			
CEFOTOXIME	100.00%	0.00%	0.00%			

Table 15 shows Enterococcus species 100% sensitive to ciprofloxacin, cefotoxime, cefuroxime and Vancomycin.

Fig. 12 List of organisms Producing ESBL

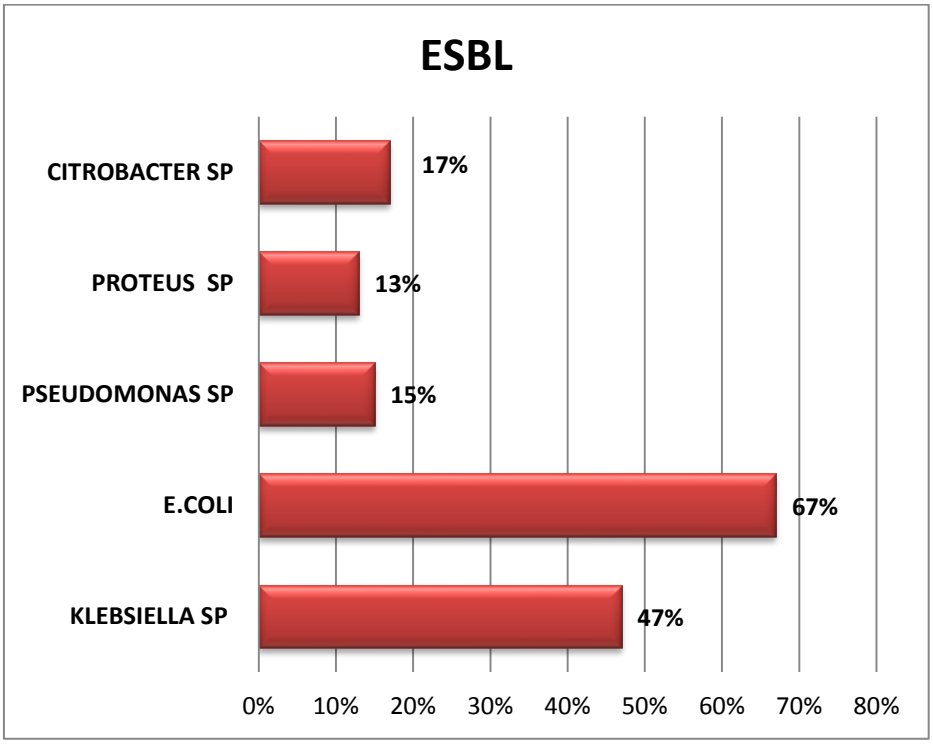


Table: 16 ESBL.

KLEBSIELLA SP	**47%**
E.COLI	**67%**
PSEUDOMONAS SP	**15%**
PROTEUS SP	**13%**
CITROBACTER SP	**17%**

Among the Gram negative bacilli 67% of E.coli and 47% of Klebsiella species were ESBL producers. (Table:16) (fig:12)

DISCUSSION

This study presents clinical and microbiological profile of Diabetic foot ulcers. About 150 to 170 million populations are suffering from diabetes mellitusworldwide.[1] In India nearly 40 million people are diabetics and their socioeconomic status is poor. Diabetic foot infections are seen in 20% of the patients and hence is the most commonly faced clinical problem. Ulcers treated in appropriately may lead to amputation or disarticulation in varying levels atleast once in such patients life time.[62]

This study was carried out at SMIMS, Kulasekharamfrom June 2014 to August 2015. Samples (swabs, aspirated pus, debrided tissue) from 75 patients of diabetic foot ulcer was collected after receiving written consent from the patient.

Majority of patients (80%) were in the age group of 51 to 70 years. Sixty patients (93.3%) were suffering from type 2 diabetes mellitus. Rani.K.L et al 2013, Kaur.N et al 2014, Zubair.M 2010 also showed that type-2 diabetes was the predominating presentation of the patients suffering from diabetic foot ulcer.[71,78,58]

Duration of diabetes mellitus is also contributing factor for development of diabetic foot ulcer as seen in our study. Fourty six (76.7%) of the 60 patients in the age group of 51 to 70 years had 6 to 15 years of the

duration of diabetes mellitus. These patients may be immunocompromised due to age factor, type of diabetes mellitus and are more prone for development of diabetes foot ulcer. The possibility of these patients ending up in amputation is high, unless preventive measures are taken up at an early stage.Although the follow up of the patients could not be done in our study. The possibility of these patients developing complications are high due to their immunocompromised state.

Majority of patients in our study had HbA1C level more than 8%, which showing poor control on diabetes mellitus. Fourty seven (78.3%) of our patients in the age group of 51 to 70 years had poor/ uncontrolled diabetes mellitus as evidenced by HBA1C levels. This also could be a contributing factor for subsequent development of complications, which may ultimately results in the amputation. Madanchi.N et al 2013,[76] had also showed the association between HBA1C levels and development of complications. This was statistically significant in our study ($p < 0.05$).

Smoking can cause microvasculitis and diminish blood flow to the affected part. (Brike.J.A et al 1992)[11]. In our study majority of patients 71% were smokers of these 46 (76.6%) were ina age group of 51 to 70 years, majority of whom had developed complications and presented with Wagners grade 3 or grade 4 ulcers.

Poorly controlled diabetes leads to accumulation of these sugar products resulting in decrease in the synthesize of nerve cell, which required for normal neuron conduction and also depletion of nicotinamide adenine dinucleotide phosphate stores. There is a result in oxidative stress on the nerve cell and increase in vasoconstriction leading to ischaemia, which will promote nerve cell injury and death (Boring.C.K 2001)[89]. Fourty two (70%) of the patients in the age group of 51 to 70 years had associated neuropathy which increased the chances of developing ulcers. This has been the observation of Mohanasoundaram.K.M et al 2012.[90]On our study there was statistical significance of diabetic foot ulcer when compared with non smokers ($p<0.05$).

Vasculopathy is one of the important contributing factor for development of diabetic foot ulcer as showed by Caputo.G.M et al 1994.[12] Sixteen (26.6%) of our patients who were in the age group of 51 to 70 had developed vasculopathy which must have contributed to the development of ulcer.

The aerobic pathogens isolated were predominantly gram negative (79) followed by gram positive (28) bacteria (fig:11). Of these107, 70 gram negative bacilli and 20 gram positive cocci were isolated in the age group between 51 to 70 years. Of 70 gram negative bacilli isolated in the age group of 51 to 70 years, Pseudomonas species was the predominant isolate followed

by Klebsiella species, Proteus species, E.coli, Citrobacter species, Acinetobacter species and Enterobacter species.

Among the gram positive cocci organisms isolated in this age group (51 to 70years), Staphylococcus aureus was the predominant pathogen followed by coagulase negative Staphylococci, Streptococci pyogenes, other beta hemolytic Streptococci, Streptococcus species and only one Enterococcus species was isolated in this age group. A evidenced by Pappu.K et al, Shanmugam.P et al, also have shown the similar findings.[61,70]

In most of the infections in the age group between 51 to 70 years it was polymicrobial (31 cases), whereas monomicrobial etiology was seen in 27 cases. As alsoreported by Chopdekar.K.A et al 2011.[60] There was no growth in two clinical samples this could be due to the prior antibiotic therapy before coming to the hospital or could be anerobic organisms the isolation of which was not attempted in the study.

The antibiogram of the isolates showed that most of the Pseudomonas species was resistant to 3rd generation Cephalosporins (74%) followed by Quinolones (76.9%) and Aminoglycosides (72%) (Table:9). Twenty five(68.4%) isolates were sensitive to Piperacillin/Tazobactam followed by Netilmicin (65.8%) and Meropenem (53.8%). Shanmugam.P 2013 et al in their study also have shown, Pseudomonas being 50% resistant to Gentamicin

and Quinolones, 61% resistant to 3rd generation Cephalosporin but 100% resistant to Meropenem. But in our study resistant to Meropenem was 46.2%.

It is suprising to note that high amount resistant to Meropenem which could be probably due to use of Carbapenems prescribed by general practitioners which must have resulted in developing resistance to Mereopenem before coming to our hospital.

Klebsiella isolates were 100% resistant to Amoxicillin. Majoritry of them were also resistant to 3rd generation Cephalosporin but 91% of the isolates were sensitive to Meropenem followed by Netilmicin(78.5%), Amikacin (69.2%), Gentamycin(50%).

Majority of Proteus species showed sensitivity to Piperacillin / Tazabactam (86.7%) followed by Meropenem (84.6%).

E.coli showed high amount of resistance to Amoxicillin, Cefotoxime, Piperacillin. However majority of orgainsms were sensitive to Netilmicin (86%), Amikacin (78%) followed by Gentamicin and Meropenem (67% each).

Citrobacter species showed 100% resistant to Amoyxicillin, Cefuroxime, Cephalexin. However they were 100% sensitive to Amikacin and Netilmicin.

Enterobacter species showed 100% sensitivity to most of the antibiotic used.

Acinetbacter species showed 100% resistant to Amoxicillin, all the 3rd generation Cephalosporins and Meropenem, Ciprofloxacin and Gentamicin. Only Amikacin and Netilmicin showed 40% sensitivity.

Among gram positive organisms Staphylococcus aureus was the predominant pathogen and the antibiogram showed 100% sensitivity to Vancomycin, Choloramphenecol and Novabiocin followed by Clindamycin (91.7%) and Teicoplanin (90%). Of the total 18 Staphylococcus aureus, 2 were MRSA. Kaur.N et al 2014,[78] also showed less sensitivity to Clindamycin is contrast to our study.

CONS showed a similar pattern of sensitivity to Vancomycin, Novobiocin, Choloramphenecol as Staphylococcus aureus. However they were less sensitive to Clindamycin (Table:13). One Coagulase negative Staphylococcus was resistant to Methicillin. Paul.S et al 2009,[56] found that 8.7% of Methicillin Resistant in their study.

All the Streptococcus pyogens were sensitive to Penicillin, they were also 100% sensitive to Netilmicin, Cefotoxime and Cefuroxime (Table:14)

Enterococcus showed 100% sensitivity to Vancomycin, Cefuroxime,Cefotoxime. Ciprofloxacin seems to be good antibiotic fortreating infections with Enterococcus species, since they showed 100% sensitivity in our study.

Extended spectrum beta lactamase producing organisms were mainly seen in E.coli (67%), Klebsiella (47%). However ESBL was not a major problem in Pseudomonas species, Proteus species, Citrobacter species. However AmpC, MBL were not looked for in our study (Table:16)(Fig:12).

SUMMARY

- This study helps the clinician to know the most common organisms causing foot ulcer infection and their sensitivity pattern.
- Polymicrobial infections was dominant than monomicrobial infections.
- Most common organisms causing diabetic foot infections were Pseudomonas species followed by Proteus species, Klebsiella species. In gram positive organism, Staphylococcus aureus was dominant followed by Streptococcus species.
- Gram negative organisms were highly sensitive to Netilmicin, Meropenem, Amikacin, Piperacillin/Tazobactam.
- Gram positive organism was highly sensitive to Vancomycin, Netilmicin, Amikacin.
- Gram negative organism was 100% resistant to Amoxicillin and highly resistant to 3rd generation Cephalosporins and Piperacillin.
- Gram positive organisms were 100% resistant to Penicillin except Streptococcus pyogenes (100% sensitivity).
- Gram negative and Gram positive organisms were highly sensitive to Netilmicin (76% and 81%). Sensitive to Amikacinwas (59% and 73%) and any one of them could be used as a monotherapy.
- Most dominant ESBL producer in our study was E.coli.

- Methicillin Resistant Staphylococcus aureus found in our study was 16.66%.
- Acinetobacter species were Multi Drug Resistant.
- Most of the diabetic ulcer patients were smokers and diabetes was poorly controlled as evidenced by HBA1C levels leading to vasculopathy and neuropathy.
- Statistical significance of <0.05 was noticed in smokers and poorly controlled diabetes mellitus as evidenced by HBA1C levels leading to neuropathy and subsequent development of foot ulcers.

CONCLUSION

- Diabetic foot ulcer infection should be treated according to culture and sensitivity report.
- To avoid unnecessary usage of antibiotic which may result in development of Multi Drug Resistant strains.
- Emprical treatment should be based on recent report of articles of same geographical region.
- Sensitivity pattern varies from place to place. This study could help clinician to know the sensitivity pattern of organism.
- Diabetic foot ulcer treatment should be based on multidisciplinary approach.
- Smokers, poor controlled diabetes mellitus patients were more prone to develop foot ulcer infection.
- It is health providers responsibility to enlighten the foot care in diabetes and consequences of foot infection and use of Proper foot wear which could decrease development of foot ulcer.

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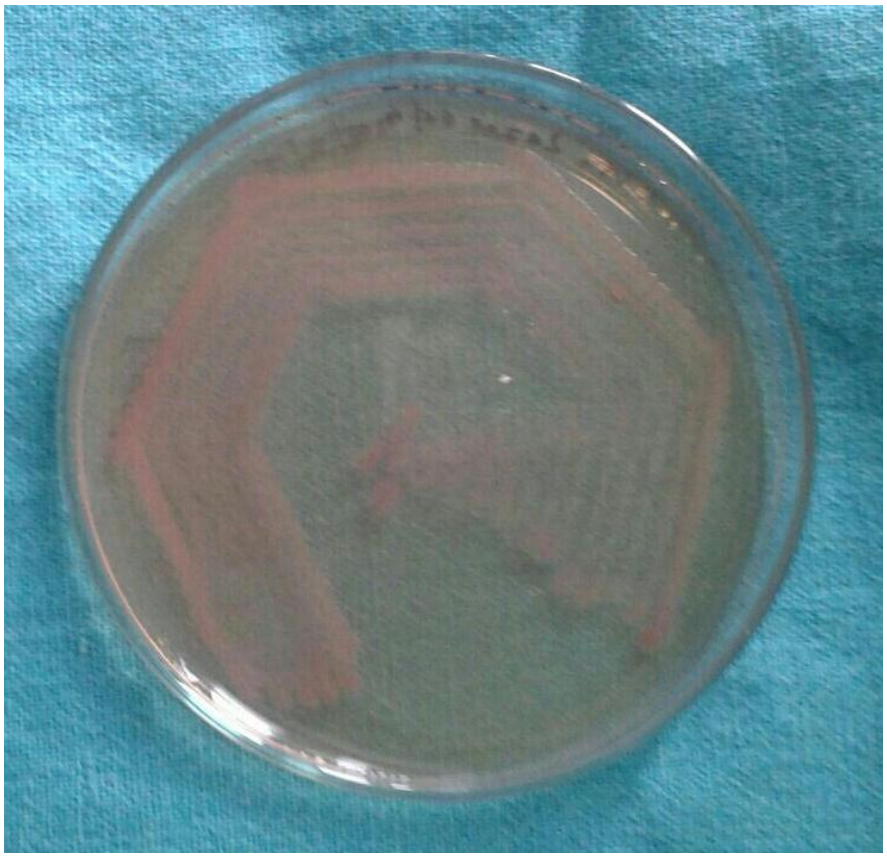
Biochemical Reactions of E.coli

Non Lactose Fermenting Colonies of Pseudomonas on Mac Conkey Agar



ANTIBIOGRAM

ESBL SCREENING

ஒப்புதல் படிவம்

பகுதி -1

ஆய்வில் பங்கேற்பாளர்களுக்கான விவரங்கள்

அன்பார்ந்த நண்பர்களே,

நீங்கள் இவ்வாராய்ச்சியில் பங்கு பெறுவதற்காக காட்டிய ஆர்வத்திற்காக உங்களுக்கு நன்றி கூறி வரவேற்கிறோம். இவ்வாராய்ச்சியில் பங்கு பெறுவதற்கு முன்னர், எதற்காக இவ்வாராய்சி நடத்தப்படுகிறது என்பதை தெரிந்துகொள்வது மிகவும் அவசியம். இப்படிவத்தின் மூலம் இவ்வாராய்ச்சியை பற்றிய விவரங்கள் மற்றும் தகவல்கள் உங்களுக்கு தெரிவிக்கப்படும். இப்படிவத்தின் மூலம் இவ்வாராய்ச்சியை பற்றியும் இவ்வாராய்ச்சி எதற்காக நடத்தப்படுகிறது என்பதை பற்றியும், இதனால் வரும் நன்மைகள், பலன்கள், ஆபத்துகள், உபாதைகள், முன்னெச்சரிக்கைகள் மற்றும் இவ்வாராய்ச்சியின் வழிமுறைகள் உங்களுக்கு விளக்கப்படும். எனவே இதனை கவனமாக படித்து பிருந்து கொள்வது மிகவும் அவசியம். இப்படிவத்தில் ஆங்காங்கே அறிவியல் துறையைச் சார்ந்த வார்த்தைகள் உபயோகப்படுத்தப்படிருக்கலாம். எனவே, உங்களுக்கு ஏதாவது சந்தேகங்களோ, அல்லது விவரங்களே தேவைப்பட்டால், இவ்வாராய்ச்சிக்கு சம்மதம், தெரிவிக்கும் முன்னரோ அல்லது இவ்வாராய்ச்சியில் இருக்கும் எந்நேரமோ, கீழ்கண்ட நேயரை தொடர்புகொண்டு சந்தேகங்களை தெளிவுபடுத்தி கொள்ளலாம்.

1, ஆராய்ச்சியாளர் ; **மருத்துவர். க. கிரீஷ்**
முதுநிலை பட்டதாரி,
நுண்ணுயிரியல் துறை,
ஸ்ரீ மூகாம்பிகா இன்ஸ்டிட்யூட் ஆப் மெடிக்கல் சயின்ஸ்,
குலசேகரம் - 629 161.

2. வழிகாட்டி : **மருத்துவர். பி.எல். உமாபதி எம்.டி**
பேராசிரியர் நுண்ணுயிரியல் துறை,
ஸ்ரீ மூகாம்பிகா இன்ஸ்டிட்யூட் ஆப் மெடிக்கல் சயின்ஸ்,
குலசேகரம் - 629 161.

3. துணை வழிகாட்டி : **மருத்துவர். யு. அருணாசலம் எம்.எஸ். எம்.சி.எச்**
தலைமை பேராசிரியர்,
அறுவை சிகிட்சை பிரிவு,
ஸ்ரீ மூகாம்பிகா இன்ஸ்டிட்யூட் ஆப் மெடிக்கல் சயின்ஸ்,
குலசேகரம் - 629 161.

4. நிறுவனத்தின் முகவரி மற்றும் விவரங்கள் :

ஸ்ரீ மூகாம்பிகா இன்ஸ்டிட்யூட் ஆப் மெடிக்கல் காம்பிளக்ஸ், படநிலம் , கன்னியாகுமரி மாவட்டம்.

5. ஆராய்ச்சியின் தலைப்பு :

ஸ்ரீ மூகாம்பிகா மருத்துவக் கல்லூரி மருத்துவமனைக்குவரும் சர்க்கரைவியாதி புண்ணிற்கு (Diabetic Foot) காரணமான பாக்டீரியாவை கண்டறியும் ஆய்வு.

6. முன்னுரை :

சர்க்கரை வியாதிக்கு இந்தியா தலைநகரம் என்றழைக்கப்படுகிறது. சர்க்கரை வியாதியினால் வரும் பின் விளைவுகளில் மிக முக்கியமான பயப்படும் படியானது சர்க்கரை வியாதிபுண். அடி படுதல், நீண்டகால சர்க்கரைவியாதி, புகைபிடிப்பது மற்றும் எலும்பு குறைபாடு ஆகிய காரணங்களாலும் புண் வரலாம்.

நரம்பு மதமதப்பு, ரத்தஓட்டம் குறைவு ஆகிய காரணங்களாலும் சர்க்கரைநோய் புண்வரலாம். தோலில் ஏற்படும் காயங்களால் நுண்ணுயிரி அதன்வழியாக சென்று உள்ளே இருக்கும் தசை நாருகளையும், சவ்வுகளையும், எலும்புகளையும் தாக்கி சர்க்கரை நோய் உள்ளவர்களுக்கு ஏற்கனவே எதிற்பு சக்தி குறைவாக இருப்பதினால் சர்க்கரை புண் வருவதற்கு வாய்பு இருக்கிறது.

7. ஆய்வின் நோக்கம் :

ஸ்ரீ மூகாம்பிகா மருத்துவமனைக்கு வரும் சர்க்கரை புண் நோயாளிகளுக்கு புண்ணிலுள்ள பாக்டீரியாவை கண்டறிதல்.

8. ஆய்வின் அறிவியல் காரணங்கள் :

சர்க்கரை வியாதி உள்ளவர்களுக்கு வரும் சர்க்கரைவியாதி புண்ணிற்கு காரணமான பாக்டீரியா பற்றிய தகவல்கள் ஸ்ரீ மூகாம்பிகா மருத்துவக்கல்லூரி மருத்துவமனையில் இல்லாததால் இந்த ஆய்வு அவசியம் ஆகிறது. பல ஆய்வுகள் இதுபற்றியிருந்தாலும் ஆராய்ச்சியின் முடிவு ஒரேமாதிரியாக இருப்பதில்லை.

9. ஆய்வின் செய்முறை :

நீங்கள் இந்த ஆய்வில் பங்கேற்பதற்குமுன் இந்த ஆய்வின் தேவைகள் அனைத்தையும் முழுமையாக புரிந்து ஒத்துக்கொள்ளவேண்டும். இந்த ஆய்வு நுண்ணுயிரியல் பிரிவுடன் இணைந்து செயல்படுகிறது. சர்க்கரை வியாதி புண் உள்ள நோயாளிகளுக்கு புண்ணிலுள்ள பழுப்பு, திசு மற்றும் 2 எம்.எல். ரத்தம் பரிசோதனைக்காக எடுக்கப்படுகிறது. இதனுடன் முதன்மையாக உங்களுடைய வயது, முகவரி மற்றும் நோயின் வரலாறு எடுக்கப்படும்.

10. பங்கேற்பாளர்களுக்கு இவ்வாராய்ச்சியினால் எதிர்பார்க்கும் அபாயங்கள் :

மிதமான அபாயங்கள் சிலசமயம் வலி, ரத்தபோக்கு ஏற்படலாம்.

11. பங்கேற்பாளர்களுக்கு இவ்வாராய்ச்சியினால் எதிர்பார்க்கப்படும் நன்மை : சர்க்கரை நோய் புண்ணிலுள்ள தொற்று எந்த கிருமியை(பாக்டீரியா) சேர்ந்தது என்பது அறியப்படுகிறது. அந்த கிருமியை அழிக்க எந்த மருந்து உதவுகிறது என்பதையும் கண்டறியப்படுகிறது, இதனால் புண் விரைவாக குணமடைந்து அதனால் ஏற்படும் பின்விளைவுகள் தடுக்கப்படுகிறது.

12. **ஆய்வின் ரகசியங்கள் :**

இவ்வாராய்ச்சியை பற்றிய தகவல்கள், சேகரிகிக்கப்பட்ட தகவல்கள் அனைத்தும் இரகசியமாக பாதுகாக்கப்படும்.

13. **இவ்வாராய்சிக்கு நீங்கள் தேர்ந்தெடுக்கப்பட்டுள்ள காரணம்:**

சர்க்கரை வியாதி புண் உள்ளதால் தேர்ந்தெடுக்கப்பட்டுள்ளேன்.

14. **எத்தனை நபர் இவ்வாராய்சியில் பங்கு கொள்வார்கள் ?** 75

15. **பங்கேற்பவர்களுக்கு நஷ்டஈடு செய்தல் (ஆய்வுக்கு சம்பந்தமான காயங்கள் ஏற்பட்டால்) :**

ஆய்வை சார்ந்த காயங்களுக்கு பங்கேற்பாளர்களுக்கு மருத்துவமனை விதிகளின்படி சிகிட்சை செய்யப்படும்.

16. **இந்த ஆய்வின் முன்னதாக பங்கேற்பாளர்களுக்கு பணம் கொடுக்கப்படுமா ?:** இல்லை

17. **நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரத்திலும் வெளிவர இயலுமா ?** ஆம்

18. **ஏதேனும் புதிய தகவல்கள் அல்லது விபரங்கள் கண்டு பிடிக்கப்பட்டால் என்னிடம் விவரம் தெரிவிக்கப்படுமா ?**

ஆம்

19. **இவ்வாராய்ச்சிக்கான கால வரையறை ?** ஒரு நாள்

20. **இவ்வாராய்ச்சியை பற்றிய இதர தகவல்கள்** எதுவும் இல்லை

21. **ஏதாவது சந்தேகத்திற்கோ, தகவலுக்கோ விவரங்களுக்கோ தொடர்பு கொள்ள வேண்டிய நபர்?**

கீழ்கண்ட நபருக்கு

டாக்டர். க. கிரீஷ்
முதுநிலை பட்டதாரி,
நுண்ணுயிரியல் பிரிவு,
ஸ்ரீ முகாம்பிகா இன்ஸ்டிட்யூட் ஆப் மெடிக்கல் சயின்ஸ்,
குலசேகரம்.
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இடம் : குலசேகரம்
தேதி : ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பவரின் கையொப்பம்

ஒப்புதல் படிவம் -2

பங்கேற்பாளரின் ஒப்புதல் படிவம்

இந்த் ஆராய்ச்சியின் தகவல்கள் அனைத்தும் என்னிடம் தெளிவாக எழுத்து மூலம் விளக்கப்பட்டுள்ளது. இந்த ஆராய்ச்சியின் முடிவுகள் எனக்கு நேரடியாக பயன் பெறாவிட்டாலும் மருத்துவத்துறையில் முன்னேற்றத்திற்கு பயன்படும் என்பதை அறிவேன். இவ்வாராய்ச்சியைப் பற்றி நான் தெளிவாக புரிந்து கொண்டுள்ளேன் மற்றும் இதைப்பற்றி என் சந்தேகங்களைத் தெளிவுபடுத்தியுள்ளேன். என்பதை அறிவேன். இதிலிருந்து எந்நேரமும் எக்காரணமுமின்றி என்னால் வெளிவர இயலும் என்பதை அறிவேன் அவ்வாறு நான் வெளிவந்தாலும் இந்த மருத்துவமனையில் எனக்கு கிடைக்கும் மருத்துவ உதவி எவ்விதத்திலும் பாதிக்கப்படாது. என்பதையும் அறிவேன். இவ்வாராய்ச்சியின் மூலம் வரும் முடிவுகள் மற்றும் தகவல்களை அறிவியல் துறையின் பயன்பாடுகளுக்கு உபயோகப்படுதித்திக்கொள்ள சம்மதிக்கிறேன். எனக்கு இவாவாராய்ச்சியைப் பற்றி விபரமான தகவல் அடங்கிய படிவம் தரப்பட்டுள்ளது. சர்க்கரைவியாதி புண்ணிற்கு (Diabetic Foot) காரணமான பாக்டீரியாவை கண்டறிவதற்கு பற்றிய படிப்பில் பங்கு கொள்வதற்கு எனக்கு முழு சம்மதம்.

பெயர்: **முகவரி :**

மருத்துவமனை எண்:

சாட்சிகள் :

பங்கேற்பவரின் கையொப்பம்

1.

2.

தேதி:
இடம்: குலசேகரம்.

ஸ்ரீ மூகாம்பிகா மருத்துவக்கல்லூரி மருத்துமனை

நுண்ணுயிரியல் துறை

தலைப்பு : சர்க்கரை நோய் புண்ணிர்க்கு காரணமான பாக்டீரியாவை கண்டறிதல்.

தேதி :

வரிசை எண் :

பெயர் :

வயது :

முகவரி :

தொலைபேசி எண் :

மருத்துவமனை எண் :

உங்களுக்கு சர்க்கரைவியாதி உள்ளதா :

எத்தனை வருடங்களாக சர்க்கரை வியாதி உள்ளது :

சர்க்கரை வியாதிக்கு சிகிட்சை எடுக்கிறீர்களா :

ரத்தக்கொதிப்பு, இருதயநோய் மற்றும் கொழுப்பு உள்ளதா :

இதற்கு முன்பு சர்க்கரைவியாதி புண் வந்துள்ளதா :

காய்ச்சல் உள்ளதா :

CONSENT FORM.

PART 1 OF 2

INFORMATION FOR PARTICIPANTS OF THE STUDY.

Dear volunteers,

We welcome you and thank you for your keen interest in participation in this research project.Before you participate in this study,it is important for you to understand why this research is being carried out.This form will provide you all the relavant details of this research.It will explain the nature,the purpose,the benefits,the risks,the discomforts,the precautions and the information about how this project will be carried out.It is important that you read and understand the contents of the form carefully.This form may contain certain scientific terms and hence,if you have any doubts or if you want more information,you are free to ask the study personnel or the contact person mentioned below before you give consent and also at any time during the entire course of the project.

1.Name of the Investigator: **Dr.K. GREESH**
Post graduate Student
Department of Microbiology
SMIMS,Kulasekharam.

2.Name of the Guide: **Dr.B.L. UMAPATHY. M.D**
Associate Professor
Department of Microbiology,
SMIMS,Kulasekharam.

3.Name of the Co- Guide: **Dr.U. ARUNACHALAM M.S, Mch**
Professor and Head
Department of Surgary
SMIMS,Kulasekharam.

4. Insitute : . Sri Mookambika Institute of Medical Sciences,
Kulasekharam,
Kanyakumari Dt,
Tamil Nadu.

5. Title of the study:

Bacteriological Profile of diabetic foot among the patient attending Sree Mookambika Institute of Medical Sciences.

6.Background information :

India has been labeled as diabetic capital of the world. Diabetic ulcer is one of the most feared complication of diabetes mellitus, many factors like trauma, duration of diabetes, smoking and deformity can cause ulcer in diabetic patients.

The major factor which predisposed to the foot ulceration, which lead to the infected was usually related to peripheral neuropathy and an in impaired circulation which limited the access of the phagocytes.

Ones the protective layer of skin is broken, the deep tissue are exposed to bacterial colonization. Infection are facilitated by immunological deficits which are related to diabetes mellitus, and they rapidly progress to the deep tissue.

7.Aims and objectives:

The study will be carry out to determine the bacterial profiles of infected ulcers and the antibiotic sensitivity pattern of the isolates.

8.Scientific justification of the study:

There is paucity of data of bacteriological profile of diabetic ulcer at Sree Mookambika Insitiute of Medical Sciences.

9. Procedure for the study.

You are required to participate in this study only if you fully understand and agree to the requirements for the same. There will be no difference in the treatment you receive, nor will treatment be withhold. Based on your decision to participate in the study. The study is done in collaboration with department of Microbiology. 75 diabetic foot patients who volunteer are included in the study. After considering inclusion and exclusion criteria. Swab or pus or debridement tissue will be collected from the diabetic foot ulcer patients. (IP &OP).

The test kit is Hi Media Gram stain and source for culture plate is Hi Media. Your address, IP number, History of Illness, will be taken initially.

10. **Expected risks for the participants** : Moderate risk, individuals may develop pain, bleeding. If any risk happens it would be treated as per hospital guidelines.

11. **Expected benefits of research for the participants :** Susceptibility of mico organisms to antibiotics, which helps early diagnosis & cure and to prevent further complication.

12. **Maintenance of Confidentiality :** All your study records will be kept confidential. Your personal identity will not be revealed in any publication or release of results. Study records will be kept indefinitely for analysis and follow up**.**

13. **Why have I been chosen to be in this study?** To detect the organisms and its susceptibility to antibiotic, Which helps early diagnosis and treatment and to avoid further complication.

14. **How many people will be in the study?** 75

15. **Agreement of Compensation to the participants (In case of a study related injury)**: Any adverse event as experienced due to the study will be treated as per hospital guidelines

16. **Anticipated prorated payment, if any, to the participant (s) of the study**:

No

17. **Can I withdraw from the study at any time during the study period?**

Yes

18. **If there is any new finding / information ,would I be informed**? Yes

19. Expected duration of the Participants participation in the study: One Day

20. Any other pertinent information: No

21. Whom do I contact for further information?

For any study related queries, you are free to contact

Dr.K. GREESH
Post graduate Student
Department of Microbiology
SMIMS,Kulasekharam.
Ph: 9566086742
Email ID : **drgreeshk@gmail.com**

Place:

Date: **Signature of principal Investigator**

Signature of the Participant.

CONSENT FORM

PART – II

The details of the study have been explained to me in writing and the details have been fully explained to me. I am aware that the results of the study may not be directly beneficial to me but will help in the advancement of medical sciences. I confirm that I have understood the study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have been given an information sheet giving details of the study. I fully consent to participate in the study titled" Bacteriological profile of diabetic foot ulcer among the patient attending Sree Mookambika Institute of Medical Sciences.

Name : **Address :**

Hospital No :

Signature of the participant

Witness

1.

2.

Date :

Place : Kulasekharam

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES,

KULASEKHARAM

DEPARTMENT OF MICROBIOLOGY

Proforma

Study Title : Bacteriological profile of diabetic foot among the patients attending Sree Mookambika Institute of Medical Sciences, Kulasekharam,

Serial No : Date:

Name :

Age :

Address :

Contact No :

Hospital Reference No:

Type of diabetes:

History of Diabetes mellitus and Treatment :

Duration of Diabetes :

History of Hypertention / IHD/Dyslipidemia :

Duration of foot ulcer:

Antibiotic treatment:

Foul smell:

Crepitation:

Purulent discharge:

Vasculopathy:

Neuropathy:

Osteomyelitis:

Cellulitis:

Gangrene:

Previous History of foot ulcer :

History of fever :

Smoking:

Alcohol:

Investigations:

Blood sugar level:

Foot X-ray if needed: